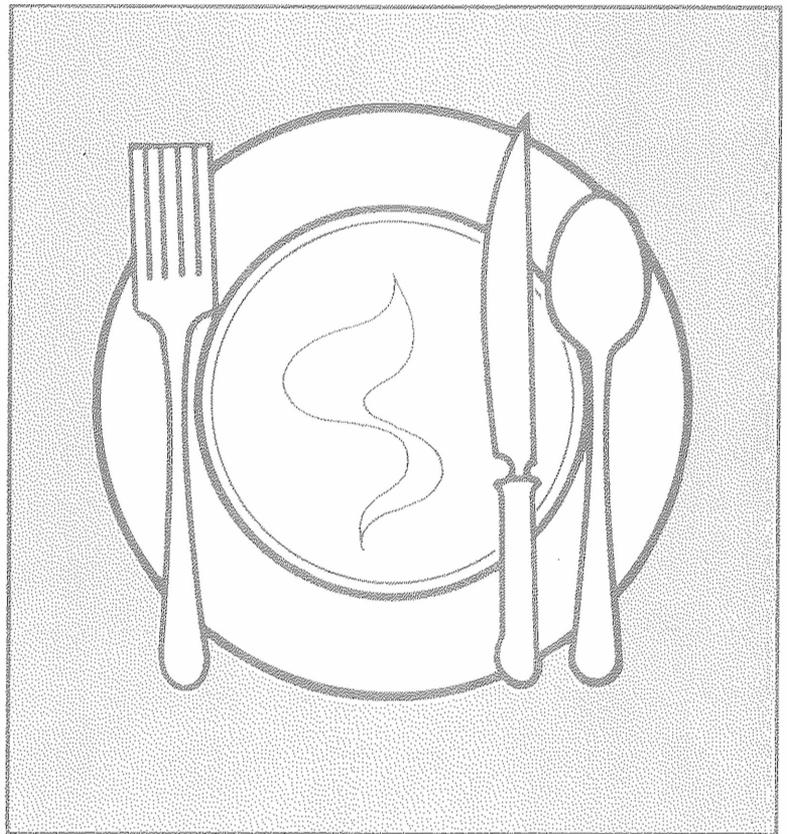


ANNUAL SUMMARY 1979

ISSUED APRIL 1981

CENTERS FOR DISEASE CONTROL
FOODBORNE DISEASE

SURVEILLANCE



PREFACE

This report summarizes information received from state and local health departments, the Food and Drug Administration, the U.S. Department of Agriculture, and private physicians. The information is preliminary and is intended primarily for use by those with responsibility for disease control activities. Anyone desiring to quote this report should contact the Enteric Diseases Branch for confirmation and further interpretation.

Contributions to the report are most welcome. Please address them to:

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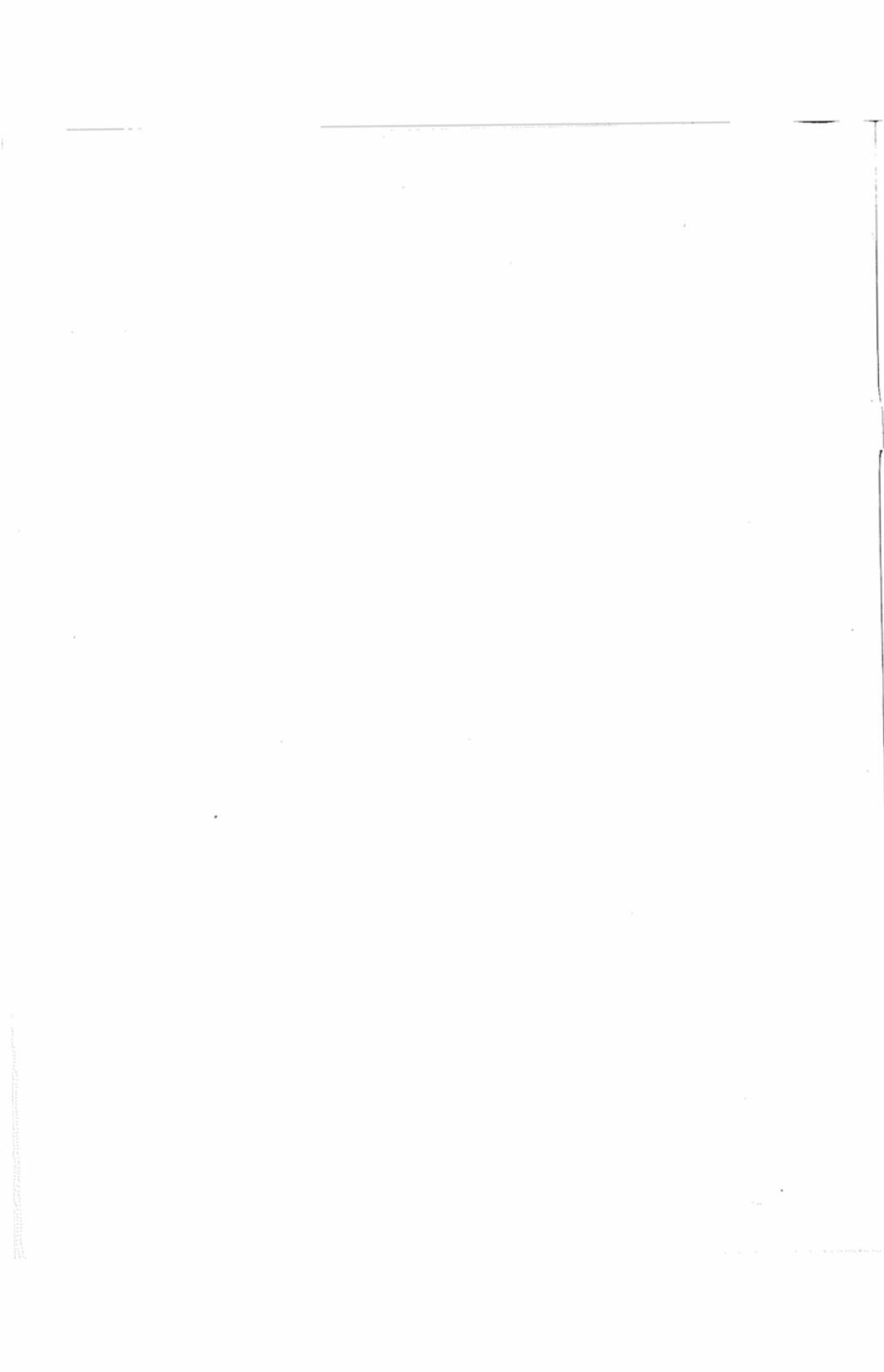
SUGGESTED CITATION

Centers for Disease Control: Foodborne Disease Outbreaks
Annual Summary 1979
Issued April 1981

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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service
Centers for Disease Control

Memorandum

Date June 30, 1981

From Enteric Diseases Branch
Bacterial Diseases Division, CID

Subject Error in Column Heading
Foodborne Disease Surveillance Report, Annual Summary 1979

To Persons receiving report

In the Foodborne Disease Surveillance Report, Annual Summary 1979, the heading of the last column in Section H (Line Listing of Foodborne Disease Outbreaks) is incorrect; the heading should read "Location where food eaten." Notes after the asterisk at the bottom of the table should be deleted.

J. Glenn Morris
J. Glenn Morris, M.D., M.P.H.&T.M.

I. SUMMARY

In 1979 there were 460 outbreaks (13,207 cases) of foodborne disease reported to the Centers for Disease Control. The etiology was confirmed in 37% of outbreaks. Bacterial pathogens accounted for 119 outbreaks (6,806 cases); salmonellae was the most frequently implicated bacterial pathogen. Chemical agents accounted for 36 outbreaks (250 cases); ciguatera poisoning was the most common chemical etiology. In 48% of outbreaks food was eaten in a restaurant. The most common contributing factor in outbreaks was improper holding temperatures.

II. INTRODUCTION

A. History

The reporting of foodborne and waterborne diseases in the United States began over half a century ago when state and territorial health officers, concerned about the high morbidity and mortality caused by typhoid fever and infantile diarrhea, recommended that cases of enteric fever be investigated and reported. The purpose was to obtain information about the role of food, milk, and water in outbreaks of intestinal illness as the basis for sound public health action. Beginning in 1923, the United States Public Health Service published summaries of outbreaks of gastrointestinal illness attributed to milk. In 1938, it added summaries of outbreaks caused by all foods. These early surveillance efforts led to the enactment of important public health measures which had a profound influence in decreasing the incidence of enteric diseases, particularly those transmitted by milk and water.

From 1951 through 1960, the National Office of Vital Statistics reviewed reports of outbreaks of foodborne illness and published summaries of them annually in Public Health Reports. In 1961 the Centers for Disease Control (CDC), then the Communicable Disease Center, assumed responsibility for publishing reports on foodborne illness. For the period 1961-66, CDC discontinued publication of annual reviews, but reported pertinent statistics and detailed individual investigations in the Morbidity and Mortality Weekly Report (MMWR).

In 1966 the present system of surveillance of foodborne and waterborne diseases began with the incorporation of all reports of enteric disease outbreaks attributed to microbial or chemical contamination of food or water into an annual summary. Since 1966 the quality of investigative reports has improved primarily as a result of more active participation by state and federal agencies in the investigation of foodborne and waterborne disease outbreaks. Due to increasing interest and activity in waterborne disease surveillance, foodborne and waterborne disease outbreaks have been reported in separate annual summaries since 1978.

B. Objectives

Foodborne disease surveillance has traditionally served 3 objectives:

1. Disease Prevention and Control: Early identification and removal of contaminated products from the commercial market, correction of faulty food preparation practices in food service establishments and in the home, and identification and appropriate treatment of human carriers of foodborne pathogens are the fundamental prevention and control measures resulting from surveillance of foodborne disease.
2. Knowledge of Disease Causation: The responsible pathogen was not identified in over 60% of foodborne disease outbreaks reported to CDC in each of the last 5 years. In many of these outbreaks pathogens known to cause foodborne illness may not have been identified because of late or incomplete laboratory investigation. In others, the responsible pathogen may have escaped detection even when a thorough laboratory investigation was carried out because the pathogen is not yet appreciated as a cause of foodborne disease or because it cannot yet be identified by available laboratory techniques. It is probable that these pathogens can be identified and suitable measures

to prevent or control diseases caused by them can be instituted if more thorough clinical, epidemiologic, and laboratory investigations are employed.

3. Administrative Guidance: The collection of data from outbreak investigations permits assessment of trends in etiologic agents and food vehicles and focuses on common errors in food handling. By compiling the data in an annual summary, it is hoped that local and state health departments and others involved in the implementation of food protection programs will be kept informed of the factors involved in foodborne disease outbreaks. Comprehensive surveillance would result in a clearer appreciation of priorities in food protection, institution of better training programs, and more rational utilization of available resources.

III. FOODBORNE DISEASE OUTBREAKS

A. Definition of Outbreak

For the purpose of this report, a foodborne disease outbreak is defined as an incident in which (1) 2 or more persons experience a similar illness, usually gastrointestinal, after ingestion of a common food, and (2) epidemiologic analysis implicates the food as the source of the illness. There are a few exceptions; 1 case of botulism or chemical poisoning constitutes an outbreak. Outbreak etiologies are classified as confirmed if specific laboratory, epidemiologic, or clinical criteria are met (Appendix A).

B. Source of Data

Outbreaks are reported to CDC on a standard reporting form (Appendix B). Reports come most frequently from state and local health departments; reports may also be received from federal agencies such as the Food and Drug Administration (FDA), U.S. Department of Agriculture (USDA), the U.S. armed forces, and occasionally from private physicians. Forms are reviewed at CDC to see if a specific etiology for the outbreak can be confirmed and, in some instances, questions about an etiologic agent may be referred back to the reporting agency. Data are otherwise accepted as reported on the forms.

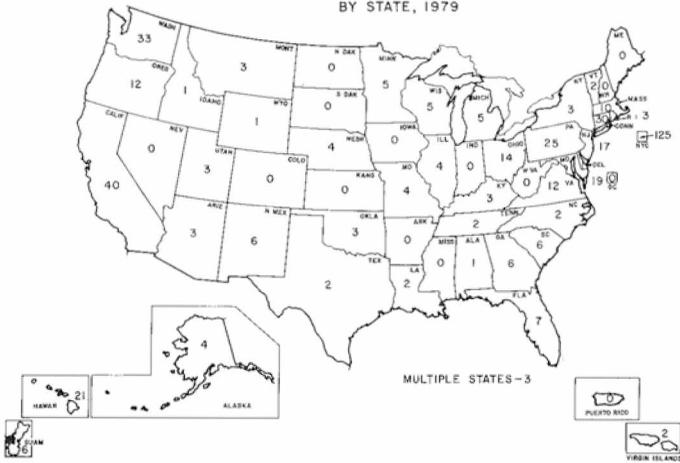
C. Interpretation of Data

The limitations on the quantity and quality of data presented here must be appreciated in order to avoid misinterpretation. The number of outbreaks of foodborne disease reported by this surveillance system clearly represents only a small fraction of the total number that occur. The likelihood of an outbreak coming to the attention of health authorities varies considerably depending on consumer and physician awareness, interest, and motivation to report the incident; for example, large outbreaks, restaurant-associated outbreaks, and outbreaks involving serious illness, hospitalizations, or deaths are more likely to come to the attention of health authorities than cases of mild illness following a family cookout. Just as this report should not be the basis of firm conclusions about the absolute incidence of foodborne disease, it should not be used to draw conclusions about the relative incidence of foodborne disease of various etiologies. For example, foodborne diseases characterized by short incubation periods such as those of chemical etiology or outbreaks caused by staphylococcal enterotoxin are more likely to be recognized as common-source foodborne disease outbreaks than those diseases with longer incubation periods, such as hepatitis A, in which there may be masking of the common-source nature of the cases. Outbreaks involving Bacillus cereus, Escherichia coli, Vibrio parahaemolyticus, Yersinia enterocolitica, or Campylobacter fetus ssp jejuni are probably less likely to be confirmed because these organisms are often not considered in clinical, epidemiologic, and laboratory investigations. Pathogens which generally cause mild illness will be under-represented, while those causing serious illness, such as Clostridium botulinum, are more likely to be identified. Similarly, restaurant-or commercial-product-associated outbreaks have a higher likelihood of being reported.

D. Analysis of Data

In 1979 there were 460 outbreaks (13,207 cases) of foodborne disease reported to CDC. Reports were received from 38 states, as well as from the U.S. Virgin Islands, Guam, and the U.S. Trust Territories of the Pacific (Figure 1). New York reported 128 outbreaks, with 125 of those from New York City; California reported the next largest

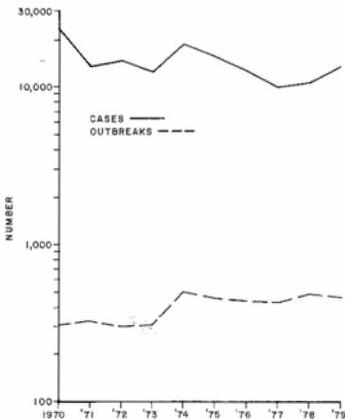
Fig. 1 OUTBREAKS OF FOODBORNE DISEASE REPORTED TO THE CENTERS FOR DISEASE CONTROL, BY STATE, 1979



number of outbreaks (40), followed by Connecticut (30). In 3 outbreaks cases were reported from multiple states. The total number of outbreaks and cases over the last 10 years is shown in Figure 2.

In 172 outbreaks (7,378 cases) an etiology was confirmed (Table 1). Bacterial pathogens accounted for 69% of confirmed outbreaks and 92% of cases. In keeping with the pattern observed during the last several years *Salmonella* was responsible for the most outbreaks (44) and the most cases (2,794); *Staphylococcus aureus* was the next most common, accounting for 34 outbreaks and 2,391 cases (Table 2). One outbreak was attributed to *Enterobacter cloacae*; >10⁵ organisms per gram were isolated from turkey and gravy served at a Thanksgiving meal, and from stools of some ill individuals. Toxicogenicity testing was not done on the isolates.

Fig. 2 NUMBER OF CASES AND OUTBREAKS OF FOODBORNE DISEASE REPORTED TO THE CENTERS FOR DISEASE CONTROL, 1970-1979



Group G *Streptococcus* was implicated in an outbreak of pharyngitis which was epidemiologically associated with consumption of chicken salad; the cook who prepared the chicken salad had a positive throat culture for group G *Streptococcus*. Five deaths were reported in association with outbreaks of *Clostridium perfringens*, with all deaths occurring in 1 large outbreak which involved a number of debilitated patients in a state mental hospital. One death was reported in association with an outbreak of *Shigella*.

Chemical etiologies accounted for 20% of the total confirmed outbreaks, but only 3% of the cases. Ciguatera poisoning was the most common etiology, accounting for 18 outbreaks and 85 cases. In 1979 *Trichinella spiralis* was the only parasitic pathogen reported, accounting for 11 outbreaks (93 cases). Viral pathogens were implicated in an additional 6 outbreaks (229 cases). The breakdown of outbreaks by etiologic category for the period 1975-1979 is shown in Table 2.

No pathogen was identified in 287 of the outbreaks (5,974 cases) reported in 1979. The extent of the investigation in these outbreaks are variable; in some instances no pathogen was identified even after an extensive laboratory investigation, while in other instances only minimal laboratory work was performed. Incubation periods were known for illnesses in 248 of the outbreaks. In 8 outbreaks the incubation period was reported as <1 hour; in 124 outbreaks the incubation period ranged between 1 and 7 hours; in 59 outbreaks the incubation period was 8 to 14 hours; while in 57 outbreaks the incubation period was >15 hours. Two deaths were reported in association with outbreaks of unknown etiology.

A number of different vehicles were implicated in the 1979 outbreaks (Table 3). The most common vehicle was beef, accounting for 20 outbreaks; the most common pathogen associated with beef was C. perfringens (7 outbreaks). Outbreaks involving ham were most often associated with Staphylococcus (8 of 10 outbreaks), with outbreaks due to other types of pork generally involving T. spiralis. With the exception of 1 case of botulism, all outbreaks associated with fish were due to either ciguatera or scombroid. Amberjack accounted for 8 of the 18 ciguatera outbreaks, while mahi-mahi (dolphin) was the most common vehicle in scombroid poisoning. No vehicle was identified in 41 of the 173 outbreaks of known etiology; 23 of these outbreaks involved Salmonella, with 50% of the Salmonella outbreaks involving an unknown vehicle. As might be expected, in 248 of the 287 outbreaks of unknown etiology, no vehicle of transmission was identified.

Two hundred twenty outbreaks were restaurant-associated, compared with 118 outbreaks associated with foods eaten at home (Table 4). Outbreaks associated with Staphylococcus aureus presented an exception to this trend, with 11 outbreaks associated with food prepared in the home compared with 4 restaurant-associated outbreaks. Outbreaks associated with C. botulinum were all associated with home-prepared foods. Outbreaks attributed to scombroid poisoning tended to occur in restaurants, and outbreaks attributed to ciguatera poisoning tended to occur at home. Outbreaks of foodborne illness occurred more frequently in the spring and fall (Table 5); 1 exception to this trend was seen with Salmonella-associated outbreaks which occurred more frequently in the summer. In 165 outbreaks the reporting agency specified a factor or factors which they felt contributed to the outbreak (Table 6). The most common factor in bacterial outbreaks was improper holding temperature, which was cited in 52 (87%) of 60 outbreaks. The next most common factor was poor personal hygiene, followed by inadequate cooking; with the exception of T. spiralis outbreaks, all of which were attributed to inadequate cooking, a similar pattern was seen with other etiologic agents.

E. Comments

It should be emphasized again that there are limitations in the quantity and quality of the data presented in this report. The variability in reporting can be seen by looking at the distribution of outbreaks by state. New York City, for example, reported 98% of the outbreaks occurring in New York State, although it accounts for less than 50% of the state's population; similarly, Connecticut reported 30 outbreaks, more than all of the southeastern states combined. While it is possible that New York City and Connecticut have an increased rate of foodborne disease, it is more likely that these differences simply represent differences in reporting. The same variability in reporting can be seen when looking at the number of outbreaks by pathogen. Our data show that C. botulinum is as common a foodborne pathogen as Shigella, a conclusion which can only be explained on the basis of more complete reporting for botulism than for shigellosis.

The number of outbreaks of foodborne disease reported to CDC per year over the last 10 years has remained relatively constant. There has been increasing variability in the number of cases reported each year, a change which can usually be explained by the occurrence of several large outbreaks involving 1,000 or more people. The distribution of cases by etiology has also remained fairly constant. Etiologies have been confirmed in 40% or less of outbreaks over the last 5 years. When the etiology has been confirmed, bacterial pathogens have consistently accounted for approximately two-thirds of outbreaks, with chemical etiologies responsible for an additional 25%. Salmonella has remained the most common bacterial foodborne pathogen, followed by S. aureus and (excluding C. botulinum) C. perfringens; there is a suggestion that C. perfringens outbreaks may be being recognized more frequently, with S. aureus outbreaks being less frequently recognized. Among chemical etiologies, ciguatera poisoning remains the most common, followed by scombroid poisoning.

E. cloacae and Streptococcus Group G were both included as foodborne pathogens for the first time in this year's report. Although neither has been clearly shown to be a foodborne pathogen, the circumstances in the outbreaks listed were such that we felt it was strongly suggestive that the organisms were the responsible pathogens. Additional work is needed to characterize these and other possible foodborne disease pathogens; non-O1 Vibrio cholerae and B. cereus, for example, have been generally accepted as pathogens only within the past decade. The large number of outbreaks in which no pathogen was identified should serve as a challenge to improve investigative skills so as to identify known pathogens more frequently, and to look for new and as yet unidentified pathogens.

Table 1
Confirmed Foodborne Disease Outbreaks, Cases, and Deaths, by Etiology,
United States, 1979

<u>Etiology</u>	<u>Number of Outbreaks</u>	<u>(%)</u>	<u>Number of Cases</u>	<u>(%)</u>	<u>No. of Deaths</u>
<u>BACTERIAL</u>					
<u>Brucella</u>	2	(1.2)	18	(0.2)	-
<u>C. botulinum</u>	7	(4.0)	9	(0.1)	-
<u>C. perfringens</u>	20	(11.6)	1,110	(15.0)	5
<u>E. cloacae</u>	1	(0.6)	37	(0.5)	-
<u>Salmonella</u>	44	(25.6)	2,794	(37.9)	-
<u>Shigella</u>	7	(4.0)	356	(4.8)	1
<u>Staphylococcus aureus</u>	34	(19.8)	2,391	(32.4)	-
<u>Streptococcus Group G</u>	1	(0.6)	73	(1.0)	-
<u>V. cholerae (non-O1)</u>	1	(0.6)	5	(0.1)	-
<u>V. parahaemolyticus</u>	2	(1.2)	14	(0.2)	-
Total	119	(69.2)	6,806	(92.3)	6
<u>CHEMICAL</u>					
Heavy metals	1	(0.6)	18	(0.2)	-
Ciguatoxin	18	(10.4)	85	(1.2)	-
Scombrototoxin	12	(6.9)	132	(1.8)	-
Mushroom poisoning	1	(0.6)	2	(0.03)	-
Other chemical	4	(2.3)	13	(0.2)	-
Total	36	(20.9)	250	(3.4)	0
<u>PARASITIC</u>					
<u>T. spiralis</u>	11	(6.4)	93	(1.3)	-
<u>VIRAL</u>					
Hepatitis (non-B)	5	(2.9)	74	(1.0)	-
Other Viral	1	(0.6)	155	(2.1)	-
Total	6	(3.5)	229	(3.1)	0
CONFIRMED TOTAL	172	(100.0)	7,378	(100.0)	6

Table 2
Confirmed Foodborne Disease Outbreaks, by Etiology,
United States, 1975-1979

Etiology	1975 (%)	1976 (%)	1977 (%)	1978 (%)	1979 (%)
BACTERIAL					
<i>A. hinshawii</i>	1 (0.5)	-	1 (0.6)	-	-
<i>B. cereus</i>	3 (1.6)	2 (1.5)	-	6 (3.9)	-
<i>Brucella</i>	-	-	-	-	2 (1.2)
<i>C. botulinum</i>	14 (7.3)	23 (17.6)	20 (12.7)	12 (7.8)	7 (4.0)
<i>C. perfringens</i>	16 (8.4)	6 (4.6)	6 (3.8)	9 (5.8)	20 (11.6)
<i>E. cloacae</i>	-	-	-	-	1 (0.6)
<i>E. coli</i>	-	-	-	1 (0.6)	-
<i>Salmonella</i>	38 (19.9)	28 (21.4)	41 (26.1)	45 (29.2)	44 (25.6)
<i>Shigella</i>	3 (1.6)	6 (4.6)	5 (3.2)	4 (2.6)	7 (4.0)
<i>Staphylococcus aureus</i>	45 (23.6)	26 (19.8)	25 (15.9)	23 (14.9)	34 (19.8)
<i>Streptococcus</i> Group D	1 (0.5)	-	-	1 (0.6)	-
<i>Streptococcus</i> Group G	-	-	-	-	1 (0.6)
<i>V. cholerae</i> O1	-	-	-	1 (0.6)	-
<i>V. cholerae</i> (non-O1)	-	-	1 (0.6)	-	1 (0.6)
<i>V. parahaemolyticus</i>	2 (1.0)	-	2 (1.3)	2 (1.3)	2 (1.2)
<i>Y. enterocolitica</i>	-	1 (0.8)	-	-	-
Other Bacterial	-	-	-	1 (0.6)	-
Total	123 (64.4)	92 (70.2)	101 (64.2)	105 (68.2)	119 (69.2)
CHEMICAL					
Heavy metals	4 (2.1)	6 (4.6)	8 (5.1)	1 (0.6)	1 (0.6)
Ciguatoxin	19 (9.9)	6 (4.6)	3 (1.9)	19 (12.3)	18 (10.4)
Scombrototoxin	6 (3.1)	2 (1.5)	13 (8.3)	7 (4.5)	12 (6.9)
Monosodium glutamate	3 (1.6)	2 (1.5)	2 (1.3)	-	-
Mushroom poisoning	5 (2.6)	1 (0.8)	5 (3.2)	1 (0.6)	1 (0.6)
Neurotrophic shellfish	-	-	-	-	-
Paralytic shellfish	-	4 (3.1)	-	4 (2.6)	-
Other Chemicals	6 (3.1)	7 (5.3)	6 (3.8)	5 (3.2)	4 (2.3)
Total	43 (22.5)	28 (21.4)	37 (25.6)	37 (24.0)	36 (20.9)
PARASITIC					
Anisakidae	1 (0.5)	-	1 (0.6)	-	-
<i>D. latum</i>	1 (0.5)	-	-	-	-
<i>T. spiralis</i>	20 (10.5)	8 (6.1)	14 (8.9)	7 (4.5)	11 (6.4)
Total	22 (11.5)	8 (6.1)	15 (9.5)	7 (4.5)	11 (6.4)
VIRAL					
Hepatitis non-B	3 (1.6)	2 (1.5)	4 (2.5)	5 (3.2)	5 (2.9)
Echo, type 4	-	1 (0.8)	-	-	-
Other Viral	-	-	-	-	1 (0.6)
Total	3 (1.6)	3 (2.3)	4 (2.5)	5 (3.2)	6 (3.5)
CONFIRMED TOTAL	191	131	157	154	172

Table 3
Foodborne Outbreaks by Specific Etiology and Vehicle of Transmission,
United States, 1979

<u>Etiology</u>	<u>Beef</u>	<u>Lamb</u>	<u>Ham</u>	<u>Pork</u>	<u>Chicken</u>	<u>Turkey</u>	<u>Other Meat</u>	<u>Shell Fish</u>	<u>Tuna</u>	<u>Amber-jack</u>	<u>Mahi-Mahi</u>	<u>Other Fish</u>	<u>Eggs</u>
<u>BACTERIAL</u>													
<u>Brucella</u>	-	-	-	-	-	-	1	-	-	-	-	1	-
<u>C. botulinum</u>	-	-	-	-	1	-	1	-	-	-	-	-	-
<u>C. perfringens</u>	7	1	1	-	-	-	-	-	-	-	-	-	-
<u>E. colosae</u>	-	-	-	-	-	-	-	-	-	-	-	-	-
<u>Salmonella</u>	4	-	1	1	3	-	1	-	-	-	-	-	2
<u>Shigella</u>	-	-	-	-	-	-	-	2	-	-	-	-	-
<u>Staphylococcus aureus</u>	2	-	8	-	1	3	1	-	-	-	-	-	-
<u>Streptococcus Group G</u>	-	-	-	-	-	-	-	-	-	-	-	-	-
<u>V. cholerae non-01</u>	-	-	-	-	-	-	-	1	-	-	-	-	-
<u>V. parahaemolyticus</u>	-	-	-	-	-	-	2	-	-	-	-	-	-
<u>Total</u>	13	1	10	1	5	3	4	5	0	0	0	1	2
<u>CHEMICAL</u>													
<u>Heavy metals</u>	-	-	-	-	-	-	-	-	-	-	-	-	-
<u>Ciguatoxin</u>	-	-	-	-	-	-	-	-	-	8	-	10	-
<u>Scombrotoxin</u>	-	-	-	-	-	-	-	-	2	-	5	5	-
<u>Mushroom poisoning</u>	-	-	-	-	-	-	-	-	-	-	-	-	-
<u>Other Chemical</u>	-	-	-	-	-	-	-	-	-	8	-	-	-
<u>Total</u>	0	0	0	0	0	0	0	0	2	8	5	15	0
<u>PARASITIC</u>													
<u>T. spiralis</u>	-	-	-	9	-	-	2	-	-	-	-	-	-
<u>VIRAL</u>													
<u>Hepatitis (non-B)</u>	-	-	-	-	-	-	-	1	-	-	-	-	-
<u>Other Viral</u>	-	-	-	-	-	-	-	-	-	-	-	-	-
<u>Total</u>	0	0	0	0	0	0	0	1	0	0	0	0	-
<u>CONFIRMED TOTAL</u>	13	1	10	10	5	3	6	6	2	8	5	16	2
<u>UNKNOWN</u>	7	0	0	0	3	0	1	3	0	0	0	0	0
<u>TOTAL</u>	20	1	10	10	8	3	7	9	2	8	5	16	2

Table 3 (Cont'd)
 Foodborne Disease Outbreaks by Specific Etiology and Vehicle of Transmission,
 United States, 1979

Cheese	Ice Cream	Other Dairy	Baked Foods	Mush-rooms	Fruits & Veg-etable	Potato Salad	Poultry, Fish, Egg Salad	Other Salad	Chi-nese Food	Mex-ican Food	Non-Dairy Bev	Multi-ple Foods	Other Foods	Un-known	Total
-	-	-	-	-	-	-	-	-	-	-	-	-	-	1	2
-	-	-	-	-	4	-	-	-	-	-	1	-	-	-	7
-	-	-	-	-	-	-	-	-	4	-	1	1	4	20	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
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0	0	0	1	1	1	0	0	0	0	0	0	2	0	4	36
-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	11
-	-	-	-	-	-	-	-	1	-	-	-	-	-	3	5
0	0	0	0	0	0	0	0	2	0	0	0	0	0	3	6
0	1	1	2	1	7	2	7	3	0	5	1	9	4	41	173
1	1	1	2	1	2	1	0	6	3	1	1	5	1	248	287
1	2	2	4	2	9	3	7	9	3	6	2	14	5	289	460

Table 4
Foodborne Disease Outbreaks, by Specific Etiology and Place Where Food was Eaten,
United States, 1979

	<u>Home</u>	<u>Restaurant</u>	<u>School</u>	<u>Picnic</u>	<u>Church</u>	<u>Camp</u>	<u>Other or Unknown</u>	<u>Total</u>
<u>BACTERIAL</u>								
<u>Brucella</u>	1	1	-	-	-	-	-	2
<u>C. botulinum</u>	7	-	-	-	-	-	-	7
<u>C. perfringens</u>	3	10	2	-	-	-	5	20
<u>E. cloacae</u>	-	-	2	-	-	-	-	1
<u>Salmonella</u>	10	16	2	-	3	1	12	44
<u>Shigella</u>	3	3	-	-	-	1	-	7
<u>Staphylococcus aureus</u>	11	4	6	-	1	-	12	34
<u>Streptococcus Group G</u>	-	-	-	-	-	-	1	1
<u>V. cholerae Non-O1</u>	1	-	-	-	-	-	-	1
<u>V. parahaemolyticus</u>	2	-	-	-	-	-	-	2
<u>Total</u>	<u>38</u>	<u>34</u>	<u>11</u>	<u>0</u>	<u>4</u>	<u>2</u>	<u>30</u>	<u>119</u>
<u>CHEMICAL</u>								
<u>Heavy metals</u>	-	1	-	-	-	-	-	1
<u>Ciguatera</u>	12	4	-	-	-	-	2	18
<u>Scombrototoxin</u>	2	8	-	-	-	-	2	12
<u>Mushroom Poisoning</u>	-	1	-	-	-	-	-	1
<u>Other Chemical</u>	1	2	1	-	-	-	-	4
<u>Total</u>	<u>15</u>	<u>16</u>	<u>1</u>	<u>0</u>	<u>0</u>	<u>0</u>	<u>4</u>	<u>36</u>
<u>PARASITIC</u>								
<u>T. spiralis</u>	11	-	-	-	-	-	-	11
<u>VIRAL</u>								
<u>Hepatitis (Non-B)</u>	1	4	-	-	-	-	-	5
<u>Other Viral</u>	-	-	-	1	-	-	-	1
<u>Total</u>	<u>1</u>	<u>4</u>	<u>0</u>	<u>1</u>	<u>0</u>	<u>0</u>	<u>0</u>	<u>6</u>
<u>CONFIRMED TOTAL</u>	<u>65</u>	<u>54</u>	<u>12</u>	<u>1</u>	<u>4</u>	<u>2</u>	<u>34</u>	<u>172</u>
<u>UNKNOWN</u>	<u>53</u>	<u>166</u>	<u>13</u>	<u>5</u>	<u>2</u>	<u>0</u>	<u>49</u>	<u>288</u>
<u>TOTAL 1979</u>	<u>118</u>	<u>220</u>	<u>25</u>	<u>6</u>	<u>6</u>	<u>2</u>	<u>83</u>	<u>460</u>

Table 5
Foodborne Disease Outbreaks by Specific Etiology and Month of Occurrence,
United States, 1979

	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Unknown	Total
<u>BACTERIAL</u>														
<u>Brucella</u>	-	-	-	-	1	1	-	-	-	-	-	-	-	2
<u>C. botulinum</u>	-	-	-	3	-	1	1	-	1	-	-	1	-	7
<u>C. perfringens</u>	4	1	4	2	3	-	-	1	1	1	2	1	-	20
<u>E. cloacae</u>	-	-	-	-	-	-	-	-	-	-	1	-	-	1
<u>Salmonella</u>	2	2	-	3	6	9	7	5	5	-	3	2	-	44
<u>Shigella</u>	-	-	-	1	3	-	1	1	-	-	1	-	-	7
<u>Staphylococcus aureus</u>	3	1	3	1	3	-	1	4	3	4	7	4	-	34
<u>Streptococcus Group G</u>	-	-	-	-	-	1	-	-	-	-	-	-	-	1
<u>V. cholerae O1</u>	-	-	-	-	-	-	-	-	-	-	1	-	-	1
<u>V. parahaemolyticus</u>	-	2	-	-	-	-	-	-	-	-	-	-	-	2
<u>Total</u>	9	6	7	10	16	12	10	11	10	5	15	8	0	119
<u>CHEMICAL</u>														
Heavy metals	1	-	-	-	-	-	-	-	-	-	-	-	-	1
Ciguatoxin	2	-	4	2	4	1	3	1	1	-	-	-	-	18
Scombrototoxin	2	-	3	-	1	-	-	1	2	1	2	-	-	12
Mushroom poisoning	-	-	-	-	-	-	-	-	-	-	-	-	1	1
Other Chemical	-	-	1	-	-	1	1	-	-	1	1	-	-	4
<u>Total</u>	5	0	8	2	5	2	4	2	3	1	3	0	1	36
<u>PARASITIC</u>														
<u>T. spiralis</u>	3	2	1	-	1	1	-	1	-	1	-	1	-	11
<u>VIRAL</u>														
Hepatitis (Non B)	1	-	1	-	-	1	-	-	-	2	-	-	-	5
Other Viral	-	-	-	-	-	1	1	-	-	-	-	-	-	1
<u>Total</u>	1	0	1	0	0	1	1	0	0	2	0	0	0	6
CONFIRMED TOTAL	18	8	17	12	22	16	15	14	13	9	18	9	1	172
UNKNOWN	28	24	33	34	19	17	24	18	12	16	32	28	3	288
TOTAL 1979	46	32	50	46	41	33	39	32	25	25	50	37	4	460

Table 6
Foodborne Disease Outbreaks by Etiology and Contributing Factors,
United States, 1979

	Number of Reported Outbreaks	Number of Outbreaks In Which Factors Reported	Improper Holding Tempera- tures	Inade- quate Cooking	Contami- nated Equipment	Food From Unsafe Source	Poor Per- sonal Hygiene	Other
<u>BACTERIAL</u>								
<u>Brucella</u>	2	1	1	1	1	-	-	-
<u>C. botulinum</u>	7	-	-	-	-	-	-	-
<u>C. perfringens</u>	20	12	12	6	-	-	2	2
<u>E. cloacae</u>	1	1	1	-	-	-	1	-
<u>Salmonella</u>	44	26	22	14	12	2	17	1
<u>Shigella</u>	7	5	2	-	-	-	4	-
<u>Staphylococcus aureus</u>	34	14	13	3	3	-	7	2
<u>Streptococcus Group G</u>	1	-	-	-	-	-	-	-
<u>V. cholerae Non-01</u>	1	-	-	-	-	-	-	-
<u>V. parahaemolyticus</u>	2	1	1	1	-	-	-	-
Total	119	60	52	25	16	2	31	5
<u>CHEMICAL</u>								
Heavy metals	1	-	-	-	-	-	-	-
Ciguatoxin	18	-	-	-	-	-	-	-
Scombrototoxin	12	2	1	-	-	1	-	2
Mushroom poisoning	1	-	-	-	-	-	-	-
Other Chemical	4	1	-	-	-	-	-	1
Total	36	3	0	0	0	0	0	1
<u>PARASITIC</u>								
<u>T. spiralis</u>	11	9	-	9	-	-	-	-
<u>VIRAL</u>								
Hepatitis (Non-B)	5	3	-	1	1	-	2	1
Other Viral	1	-	-	-	-	-	-	-
Total	6	3	0	1	1	0	2	1
CONFIRMED TOTAL	172	75	53	35	17	3	33	9
UNKNOWN	288	90	62	18	34	4	37	15
TOTAL 1979	460	165	115	53	51	7	70	24

F. Guidelines for Confirmation of Foodborne Disease Outbreak

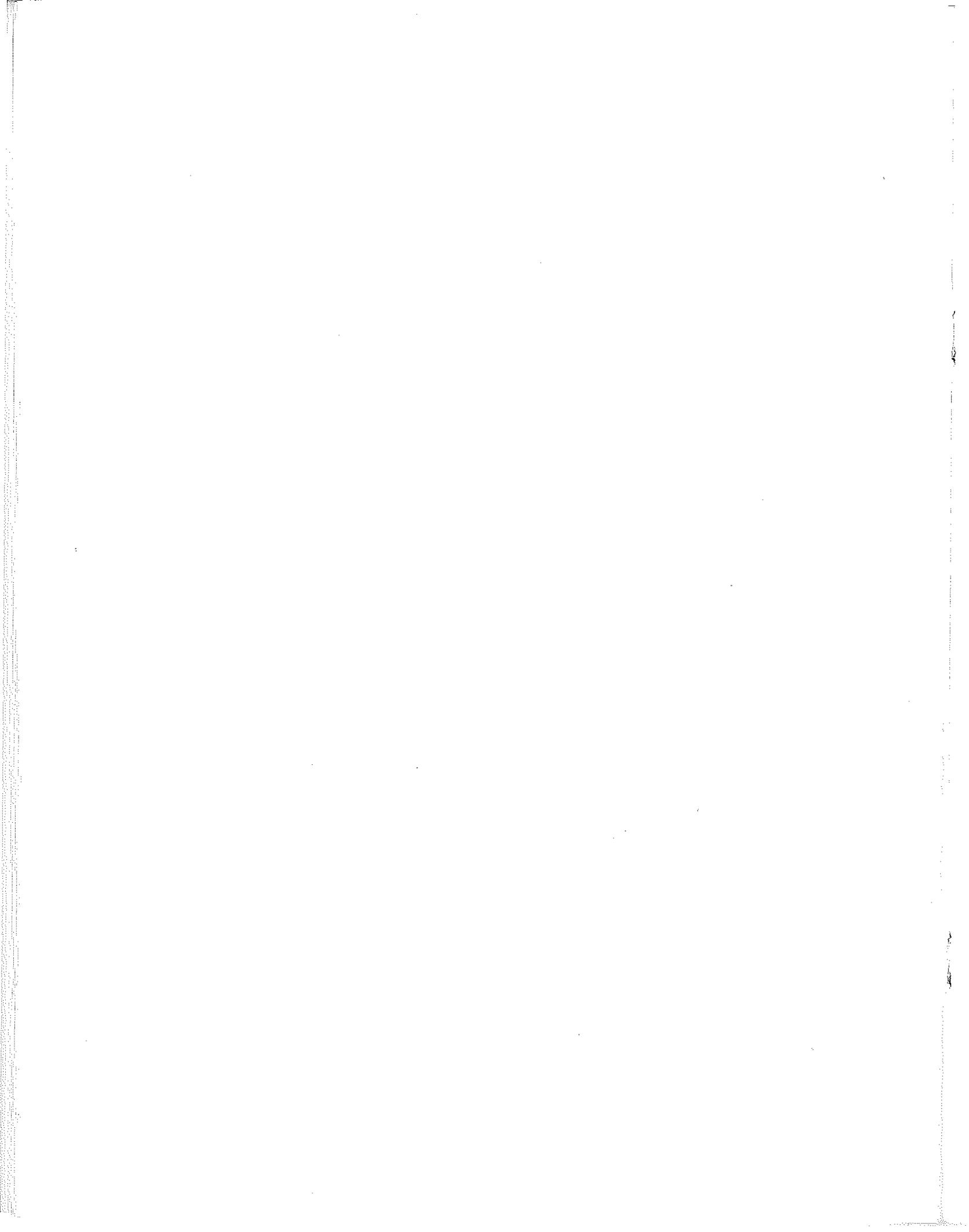
<u>BACTERIAL</u>	<u>Clinical Syndrome</u>	<u>Laboratory, clinical, and/or epidemiologic criteria for confirmation</u>
1. <u>Bacillus cereus</u>	<p>Vomiting toxin: a) incubation period 1-6 hrs. b) vomiting, some cases with diarrhea</p> <p>Diarrheal toxin: a) incubation period 6-24 hrs. b) diarrhea, abdominal cramps, some cases with vomiting</p>	<p>a) isolation of $\geq 10^5$ organisms per gram in epidemiologically incriminated food OR b) isolation of organism from stools of ill persons and not in stools of controls</p>
2. <u>Brucella</u>	<p>a) incubation period several days to several months</p> <p>b) clinical syndrome compatible with brucellosis</p>	<p>a) 4-fold increase in titer OR b) positive blood culture</p>
3. <u>Clostridium botulinum</u>	<p>a) incubation 2 hours-8 days, usually 12-48 hours</p> <p>b) clinical syndrome compatible with botulism (see CDC Botulism Manual)</p>	<p>a) detection of botulin toxin in human sera, feces, or food OR b) isolation of <u>C. botulinum</u> organism from stools OR c) clinical syndrome in persons known to have consumed same food as other individuals with laboratory-proven cases</p>
4. <u>Clostridium perfringens</u>	<p>a) incubation period 9-15 hrs.</p> <p>b) lower intestinal syndrome--majority of cases with diarrhea but little vomiting or fever</p>	<p>a) organisms of same serotype in epidemiologically incriminated food and stool of ill individuals. OR b) isolation of organisms with same serotype in stool of most ill individuals and not in stool of controls OR c) $\geq 10^5$ organisms per gram in epidemiologically incriminated food provided specimen properly handled</p>
5. <u>Escherichia coli</u>	<p>a) incubation period 6-36 hrs.</p> <p>b) gastrointestinal syndrome--majority of cases with diarrhea</p>	<p>a) demonstration of organisms of same serotype in epidemiologically incriminated food and stool of ill individuals and not in stool of controls OR b) isolation from stool of most ill individuals, organisms of the same serotype which have been shown to be enterotoxigenic or invasive by special laboratory techniques</p>

	<u>Clinical Syndrome</u>	<u>Laboratory, clinical, and/or epidemiologic criteria for confirmation</u>
6. <u>Salmonella</u>	a) incubation period 6-48 hrs. b) gastrointestinal syndrome-- majority of cases with diarrhea	a) isolation of <u>Salmonella</u> or- ganism from epidemiologically implicated food OR b) isolation of <u>Salmonella</u> organism from stools of ill individuals
7. <u>Shigella</u>	a) incubation period 12-50 hours b) gastrointestinal syndrome-- majority of cases with diarrhea	a) isolation of <u>Shigella</u> organ- ism from epidemiologically im- plicated food OR b) isolation of <u>Shigella</u> organ- ism from stools of ill individ- uals
8. <u>Staphylococcus aureus</u>	a) incubation period 30 min.- 8 hours (usually 2-4 hrs.) b) gastrointestinal syndrome-- majority of cases with vomiting	a) detection of enterotoxin in epidemiologically implicated food OR b) organisms with same phage type in stools or vomitus of ill individuals; isolation from epidemiologically impli- cated food and/or skin or nose of food handler is supportive evidence OR c) isolation of $>10^5$ organ- isms per gram in epidemiologi- cally implicated food
9. <u>Streptococcus Group A</u>	a) incubation period 1-4 days b) febrile URI syndrome	a) isolation of organisms with same M and T type from impli- cated food OR b) isolation of organisms with same M and T type from throats of ill individuals
10. <u>Vibrio cholerae 01</u>	a) incubation period 1-5 days b) gastrointestinal syndrome-- majority of cases with diarrhea and without fever	a) isolation of <u>V. cholerae 01</u> from epidemiologically incrim- inated food OR b) isolation of organisms from stools or vomitus of ill individuals OR c) significant rise in vibriocidal, bacterial agglutinating or antitoxin antibodies in acute and early convalescent sera, or significant fall in vibriocidal antibodies in early and late convalescent sera in persons not recently immunized

	<u>Clinical Syndrome</u>	<u>Laboratory, clinical, and/or epidemiologic criteria for confirmation</u>
<u>Vibrio cholerae</u> Non-01	a) incubation period up to 3 days b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of non-01 <u>V. cholerae</u> of same serotype from stools of ill persons; isolation from epidemiologically implicated food is supportive evidence
11. <u>Vibrio parahaemolyticus</u>	a) incubation period 4-30 hrs. b) gastrointestinal syndrome--majority of cases with diarrhea	a) isolation of $>10^5$ organisms from epidemiologically implicated food (usually seafood) <u>OR</u> b) isolation of Kanagawa-positive organisms of same serotype from stool of ill individuals
12. Others	clinical data appraised in individual circumstances	laboratory data appraised in individual circumstances
<u>CHEMICAL</u>		
1. Heavy metals Antimony Cadmium Copper Iron Tin Zinc, etc	a) incubation period 5 min. to 8 hrs. (usually less than 1 hr) b) clinical syndrome compatible with heavy metal poisoning--usually gastrointestinal syndrome and often metallic taste	demonstration of high concentration of metallic ion in epidemiologically incriminated food or beverage
2. Ichthyosarcotoxin		
Ciguatoxin	a) incubation period 1-48 hrs. (usually 2-8 hrs.) b) Usually gastrointestinal symptoms followed by neurologic manifestations, including paresthesia of lips, tongue, throat or extremities, and reversal of hot and cold sensation	a) demonstration of ciguatoxin in epidemiologically incriminated fish <u>OR</u> b) clinical syndrome in person(s) who have eaten a type of fish previously associated with ciguatera fish poisoning (e.g., snapper, grouper)
Puffer fish (tetrodotoxin)	a) incubation period 10 min. to 3 hrs. (usually 10-45 min.) b) paresthesia of lips, tongue, face or extremities often followed by numbness, loss of proprioception or a "floating" sensation	a) demonstration of tetrodotoxin in fish <u>OR</u> b) puffer fish epidemiologically incriminated

	<u>Clinical Syndrome</u>	<u>Laboratory, clinical, and/or epidemiologic criteria for confirmation</u>
Scombrototoxin	<p>a) incubation period 1 min. to 3 hours (usually less than 1 hour)</p> <p>b) flushing, headache, dizziness, burning of mouth and throat, upper and lower gastrointestinal symptoms, urticaria and generalized pruritus</p>	<p>a) demonstration of elevated histamine levels in epidemiologically incriminated fish</p> <p style="text-align: center;">OR</p> <p>b) clinical syndrome in person(s) known to have eaten a fish of order Scombrodel or a type of fish previously associated with scombroid poisoning (e.g., mahi-mahi)</p>
3. Monosodium glutamate	<p>a) incubation period 3 min. to 2 hours (usually less than 1 hour)</p> <p>b) burning sensations in chest, neck, abdomen or extremities, sensations of lightness and pressure over face, or a heavy feeling in the chest</p>	<p>history of large amounts (usually >1.5 grams) of MSG having been added to epidemiologically incriminated food</p>
4. Mushroom poison		
Group containing ibotenic acid and muscimol	<p>a) incubation period 1-12 hrs. (usually less than 4 hrs.)</p> <p>b) clinical syndrome compatible with mushroom poisoning by this group--often including confusion, delirium, visual disturbances</p>	<p>a) demonstration of toxic chemical in epidemiologically incriminated mushrooms</p> <p style="text-align: center;">OR</p> <p>b) epidemiologically incriminated mushrooms identified as a toxic type</p>
Group containing amanitotoxins and phallotoxins, or gyromitrin	<p>a) incubation period 5-18 hrs.</p> <p>b) characteristic clinical syndrome compatible with mushroom poisoning by this group--upper and lower gastrointestinal symptoms followed by hepatic and/or renal failure</p>	<p>a) demonstration of toxic chemical in epidemiologically incriminated mushrooms</p> <p style="text-align: center;">OR</p> <p>b) epidemiologically incriminated mushrooms identified as a toxic type</p>
Groups containing muscarine, psilocybin and psilocin, gastrointestinal irritants, disulfiram-like compounds	<p>a) characteristic incubation period</p> <p>b) clinical syndrome compatible with mushroom poisoning by these groups</p>	<p>a) demonstration of toxic chemical in epidemiologically incriminated mushrooms</p> <p style="text-align: center;">OR</p> <p>b) epidemiologically incriminated mushroom identified as toxic type</p>

	<u>Clinical Syndrome</u>	<u>Laboratory, clinical, and/or epidemiologic criteria for confirmation</u>
5. Paralytic or neurotoxic shellfish poison	a) incubation period 30 min. to 3 hours b) paresthesias of lips, mouth or face, and extremities; weakness, including respiratory difficulty in most severe cases; upper and lower gastrointestinal symptoms in some cases	a) detection of toxin in epidemiologically incriminated mollusks <u>OR</u> b) detection of large numbers of shellfish poisoning-associated species of dinoflagellates in water from which epidemiologically incriminated mollusks gathered
6. Other chemical	clinical data appraised in individual circumstances	laboratory data appraised in individual circumstances
<u>PARASITIC AND VIRAL</u>		
1. <u>Trichinella spiralis</u>	a) incubation period 3-30 days b) clinical syndrome compatible with trichinosis--often including fever, high eosinophil count, orbital edema, myalgia	a) muscle biopsy from ill individual <u>OR</u> b) serological tests <u>OR</u> c) demonstration of larvae in incriminated food
2. Hepatitis A	a) incubation period 10-45 days b) clinical syndrome compatible with hepatitis--usually including jaundice, GI symptoms, dark urine	liver function tests compatible with hepatitis in affected persons who consumed the epidemiologically incriminated food
3. Others	clinical evidence appraised in individual circumstances	laboratory evidence appraised in individual circumstances



H. Line Listing of Foodborne Disease Outbreaks

1979

H. LINE LISTING OF FOODBORNE DISEASE OUTBREAKS, 1979

Etiology	State	Number of Cases	Date of Onset	Lab Data			Location Where Food Mishandled and Eaten
				Patient	Vehicle	Food-Handler	
BACTERIAL							
BRUCELLA							
<u>Brucella</u>	California	5	6/21		+		Other or not specified meat
<u>Brucella</u>	Connecticut	13	5/16		+	+	Unknown
CLOSTRIDIUM BOTULINUM							
<u>C. botulinum</u>	Alaska	1	6/2	+	+		Other or not specified meat
<u>C. botulinum</u>	Alaska	1	9/8	+	+		Other fish
<u>C. botulinum</u>	California	1	7/5	+	+		Other vegetables
<u>C. botulinum</u>	Kentucky	2	4/8	+	+		Other vegetables
<u>C. botulinum</u>	Nebraska	1	4/24	+	+		Non-dairy beverages
<u>C. botulinum</u>	Washington	1	4/10	+	+		Other vegetables
<u>C. botulinum</u>	Washington	1	12/8	+	+		Other vegetables
CLOSTRIDIUM PERFRINGENS							
<u>C. perfringens</u>	Arizona	21	5/04		+		Beef
<u>C. perfringens</u>	California	45	5/06	+	+		Beef
<u>C. perfringens</u>	California	120	5/24		+		Unknown
<u>C. perfringens</u>	California	9	11/05		+		Beef
<u>C. perfringens</u>	California	8	12/13		+		Beef
<u>C. perfringens</u>	Georgia	5	1/28		+		Unknown
<u>C. perfringens</u>	Hawaii	25	11/23		+		Ham
<u>C. perfringens</u>	Minnesota	70	9/12	+	+		Other, not specified
<u>C. perfringens</u>	Montana	46	4/04		+		Mexican food
<u>C. perfringens</u>	New Jersey	133	10/28	+			Chicken
<u>C. perfringens</u>	South Carolina	79	8/29		+		Multiple vehicles
<u>C. perfringens</u>	Virginia	81	4/07		+	+	Lamb
<u>C. perfringens</u>	Washington	2	1/08	+			Mexican food
<u>C. perfringens</u>	Washington	2	1/24	+			Beef
<u>C. perfringens</u>	Washington	391	1/27	+			Mexican food
<u>C. perfringens</u>	Washington	4	2/26	+	+		Unknown
<u>C. perfringens</u>	Washington	4	3/07				Beef
<u>C. perfringens</u>	Washington	3	3/13		+		Beef
<u>C. perfringens</u>	Washington	2	3/13	+	+		Mexican food
<u>C. perfringens</u>	Wisconsin	60	3/05	+			Unknown
ENTEROBACTER							
<u>E. cloacae</u>	Ohio	37	11/22	+	+	+	Multiple vehicles
SALMONELLA							
<u>S. saint-paul</u>	California	52	4/14	+		+	Unknown
<u>S. thompson</u>	California	8	9/23	+		+	Eggs
<u>S. enteritidis</u>	Connecticut	72	7/16			+	Chicken salad
<u>S. enteritidis</u>	Connecticut	25	8/19	+		+	Unknown
<u>S. heidelberg</u>	Connecticut	20	9/08	+			Unknown
<u>S. typhimurium</u>	Connecticut	12	9/19	+		+	Unknown
<u>S. (undefined)</u>	Florida	108	2/17		+		Unknown
<u>S. enteritidis</u>	Georgia	20	8/10	+		+	Unknown
<u>S. infantis</u>	Georgia	998	11/04	+	+		Pork
<u>S. enteritidis</u>	Idaho	28	7/14	+	+	+	Other or not specified meat
<u>S. newport</u>	Illinois	42	6/09	+			Beef
<u>S. enteritidis</u>	Illinois	45	6/19	+	+	+	Deviled Eggs
<u>S. oranienburg</u>	Illinois	18	6/?		+		Other fruit
<u>S. litchfield</u>	Maryland	23	5/22	+		+	Unknown
<u>S. typhimurium</u>	Maryland	24	6/13	+		+	Beef
<u>S. (undefined)</u>	Maryland	11	7/14	+	+		Unknown
<u>S. enteritidis</u>	Massachusetts	15	5/13	+		+	Unknown
<u>S. typhimurium</u>	Massachusetts	200	5/23	+	+	+	Unknown
<u>S. (undefined)</u>	Massachusetts	18	6/25	+			Unknown
<u>S. enteritidis</u>	Massachusetts	100	7/14	+		+	Unknown
<u>S. enteritidis</u>	Massachusetts	164	7/23	+		+	Unknown
<u>S. enteritidis</u>	Massachusetts	55	8/15	+		+	Unknown
<u>S. typhimurium</u>	Michigan	8	5/06	+			Unknown
<u>S. (undefined)</u>	Michigan	35	9/01	+	+	+	Ham
<u>S. enteritidis</u>	Missouri	60	8/05	+	+		Unknown
<u>S. enteritidis</u>	Montana	12	4/10	+			Unknown
<u>S. heidelberg</u>	New Jersey	100	6/?	+	+	+	Other or not specified baked goods
<u>S. norwalk</u>	New Jersey	63	12/06	+		+	Unknown
<u>S. typhimurium</u>	Oregon	3	1/15		+		Other or not specified dairy products

*(A)—Food processing establishment; (B)—Food service establishment; (C)—Home; (D)—Unknown; (E)—Not applicable

Etiology	State	Number of Cases	Date of Onset	Lab Data			Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
				Patient	Vehicle	Food-Handler			
<u>S. san diego</u>	Oregon	14	2/28	+			+	Chicken salad	Restaurant
<u>S. (undefined)</u>	Oregon	10	4/12					Beef	Restaurant
<u>S. typhimurium</u>	Pennsylvania	15	6/10	+				Beef	Other
<u>S. oranienburg</u>	Pennsylvania	39	11/08	+	+			Unknown	Church
<u>S. typhimurium</u>	Pennsylvania	64	11/09	+	+			Multiple vehicles	Other
<u>S. newport</u>	South Carolina	14	7/29	+			+	Green beans	Restaurant
<u>S. (undefined)</u>	Texas	12	7/22	+			+	Chicken	Church
<u>S. newport</u>	Utah	10	1/?	+	+			Ice cream	Home
<u>S. enteritidis</u>	Vermont	24	5/17	+	+		+	Multiple vehicles	School
<u>S. typhimurium</u>	Washington	3	5/21	+				Unknown	Restaurant
<u>S. saint-paul</u>	Wisconsin	19	9/16	+				Chicken	Church
<u>S. (undefined)</u>	Guam	5	6/26	+				Unknown	Other
<u>S. oranienburg</u>	Guam	4	8/05	+				Chicken	Other
<u>S. infantis</u>	New York City	3	6/20	+	+			Unknown	Restaurant
<u>S. enteritidis</u>	New York City	17	12/25	+	+		+	Unknown	Home

SHIGELLA

<u>S. flexneria</u>	Arizona	26	5/06	+				Shellfish	Camp
<u>S. (undefined)</u>	California	11	5/03	+				Unknown	Restaurant
<u>S. sonnei</u>	Connecticut	5	7/09	+				Shellfish	Home
<u>S. flexneria</u>	Massachusetts	8	11/25	+				Unknown	Home
<u>S. sonnei</u>	Montana	13	4/13	+				Mexican food	Restaurant
<u>S. sonnei</u>	Pennsylvania	280	5/17	+			+	Fish salad	Cafeteria
<u>S. sonnei</u>	Wisconsin	13	8/25	+				Unknown	Home

STAPHYLOCOCCUS

<u>S. aureus</u>	California	245	8/13	+			+	Ham	Other
<u>S. aureus</u>	Delaware	64	3/10			+		Chicken salad	Home
<u>S. aureus</u>	Florida	28	12/24			+		Other, not specified	Other
<u>S. aureus</u>	Georgia	2	5/28			+		Ham	Home
<u>S. aureus</u>	Georgia	2	9/21			+		Unknown	Restaurant
<u>S. aureus</u>	Georgia	17	11/23			+		Ham	Home
<u>S. aureus</u>	Hawaii	6	12/28			+	+	Chicken	Home
<u>S. aureus</u>	Kentucky	239	11/12	+	+		+	Beef	School
<u>S. aureus</u>	Kentucky	10	12/25			+		Unknown	Home
<u>S. aureus</u>	Maryland	30	1/20	+		+		Macaroni salad	Home
<u>S. aureus</u>	Maryland	30	9/02			+		Unknown	Other
<u>S. aureus</u>	Massachusetts	73	12/20				+	Beef	Other
<u>S. aureus</u>	Minnesota	50	8/11			+		Egg salad	Other
<u>S. aureus</u>	Missouri	27	10/18	+	+			Ham	Restaurant

<u>S. aureus</u>	Nebraska	26	10/20			+		Multiple vehicles	Other
<u>S. aureus</u>	Nebraska	18	10/21			+		Ham	Home
<u>S. aureus</u>	New Mexico	2	1/08					Unknown	Other
<u>S. aureus</u>	North Carolina	309	7/07			+	+	Unknown	Other
<u>S. aureus</u>	Ohio	15	4/15	+	+		+	Potato salad	Home
<u>S. aureus</u>	Oklahoma	239	11/12	+	+		+	Multiple vehicles	School
<u>S. aureus</u>	Oklahoma	298	11/22	+	+		+	Turkey	Other
<u>S. aureus</u>	Oregon	3	3/07	+				Multiple vehicles	Home
<u>S. aureus</u>	Oregon	10	5/10			+	+	Multiple vehicles	School
<u>S. aureus</u>	Oregon	117	11/30	+			+	Multiple vehicles	School
<u>S. aureus</u>	Pennsylvania	130	3/06			+	+	Unknown	School
<u>S. aureus</u>	Pennsylvania	58	8/25	+	+		+	Ham	Other
<u>S. aureus</u>	Rhode Island	80	9/13	+	+			Chicken salad	Other
<u>S. aureus</u>	Texas	13	10/15			+		Potato salad	Restaurant
<u>S. aureus</u>	Vermont	33	5/27	+			+	Unknown	Other
<u>S. aureus</u>	Virginia	21	8/19	+				Ham	Church
<u>S. aureus</u>	Virginia	9	11/22				+	Turkey	Home
<u>S. aureus</u>	Washington	5	2/10			+		Ham	Home
<u>S. aureus</u>	Wyoming	163	11/16	+	+		+	Turkey	School
<u>S. aureus</u>		19	1/01			+		Other or not specified meat	Delicatessen

STREPTOCOCCUS

Group G Strep	Florida	73	6/21	+			+	Chicken salad	Other
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VIBRIOS

<u>V. cholerae non-01</u>	Florida	5	11/08	+				Shellfish	Home
<u>V. parahaemolyticus</u>	Guam	3	2/04					Shellfish	Home
<u>V. parahaemolyticus</u>	Guam	11	2/05			+		Shellfish	Home

PARASITIC

TRICHINELLA SPIRALIS

<u>T. spiralis</u>	Alaska	26	6/?			+		Other or not specified meat	Home
<u>T. spiralis</u>	Louisiana	19	2/05	+	+			Pork	Home
<u>T. spiralis</u>	Maryland	3	5/06			+		Pork	Home
<u>T. spiralis</u>	Massachusetts	6	1/16			+		Pork	Home
<u>T. spiralis</u>	New Jersey	4	3/22			+		Pork	Home

* (A)—Food processing establishment; (B)—Food service establishment; (C)—Home; (D)—Unknown; (E)—Not applicable

Etiology	State	Number of Cases	Date of Onset	Lab Data			Food-Handler	Vehicle	Location Where Food Mishandled and Eaten
				Patient	Vehicle	Handler			
<u>T. spiralis</u>	New Jersey	5	8/14		+		Pork	Home	
<u>T. spiralis</u>	New Jersey	7	10/28		+		Pork	Home	
<u>T. spiralis</u>	Pennsylvania	5	12/31		+		Pork	Home	
<u>T. spiralis</u>	Rhode Island	3	1/15		+		Pork	Home	
<u>T. spiralis</u>	Virginia	2	2/01		+		Pork	Home	
<u>T. spiralis</u>		13	1/?	+	+		Other or not specified meat	Home	

VIRAL

Hepatitis (non-B)	Alaska	14	3/13	+			Jello salad	Home
Hepatitis (non-B)	New Jersey	24	6/10	+			Unknown	Restaurant
Hepatitis (non-B)	New Jersey	24	10/01	+			Unknown	Restaurant
Hepatitis (non-B)	Pennsylvania	4	1/15	+			Unknown	Restaurant
Hepatitis (non-B)		8	10/?				Shellfish	Restaurant
Other viral	Connecticut	155	7/30				Macaroni salad	Picnic

CHEMICAL

Mushroom poison	New York City	2	??		+		Mushrooms	Restaurant
Scombrotoxin	California	3	9/26				Other fish	Home
Scombrotoxin	California	17	9/28		+		Mahi-Mahi	Restaurant
Scombrotoxin	Connecticut	12	1/16				Other fish	Restaurant
Scombrotoxin	Connecticut	14	8/01				Other fish	Restaurant
Scombrotoxin	Hawaii	14	3/16		+		Mahi-Mahi	Restaurant
Scombrotoxin	Hawaii	3	11/04		+		Other fish	Home
Scombrotoxin	Minnesota	24	1/25				Mahi-Mahi	Other
Scombrotoxin	New Jersey	35	10/04		+		Tuna	Other
Scombrotoxin	New Jersey	2	11/?				Other fish	Restaurant
Scombrotoxin	Washington	1	3/21		+		Mahi-Mahi	Restaurant
Scombrotoxin	Washington	2	5/15		+		Mahi-Mahi	Restaurant
Scombrotoxin	Virgin Islands	5	3/06				Tuna	Restaurant
Ciguatoxin	Florida	10	7/04		+		Other fish	Other
Ciguatoxin	Hawaii	2	1/02				Amberjack	Home
Ciguatoxin	Hawaii	7	1/28				Other fish	Home
Ciguatoxin	Hawaii	3	3/01				Amberjack	Home
Ciguatoxin	Hawaii	3	3/02				Amberjack	Home
Ciguatoxin	Hawaii	3	3/04				Amberjack	Home
Ciguatoxin	Hawaii	3	4/18				Other fish	Home
Ciguatoxin	Hawaii	4	4/26				Other fish	Home
Ciguatoxin	Hawaii	3	5/01				Amberjack	Home
Ciguatoxin	Hawaii	11	5/01				Amberjack	Home

Ciguatoxin	Hawaii	3	5/05				Other fish	Home
Ciguatoxin	Hawaii	3	5/29				Amberjack	Home
Ciguatoxin	Hawaii	1	6/22		+		Other fish	Other
Ciguatoxin	Hawaii	6	7/11				Other fish	Restaurant
Ciguatoxin	Hawaii	60	7/22		+		Amberjack	Restaurant
Ciguatoxin	Hawaii	3	8/27		+		Other fish	Restaurant
Ciguatoxin	Hawaii	4	9/02				Other fish	Home
Ciguatoxin	Virgin Islands	10	3/?				Other fish	Restaurant
Metal	New York City	18	1/24		+		Carbonated drink	Restaurant
Other chemical	California	5	7/18				Other, not specified	Restaurant
Other chemical	Connecticut	3	11/08		+		Other vegetables	School
Other chemical	New Jersey	3	6/02		+		Other or not specified baked goods	Home
Other chemical	Washington	2	3/31				Other, not specified	Restaurant

*(A)--Food processing establishment; (B)--Food service establishment; (C)--Home; (D)--Unknown; (E)--Not applicable

UNKNOWN

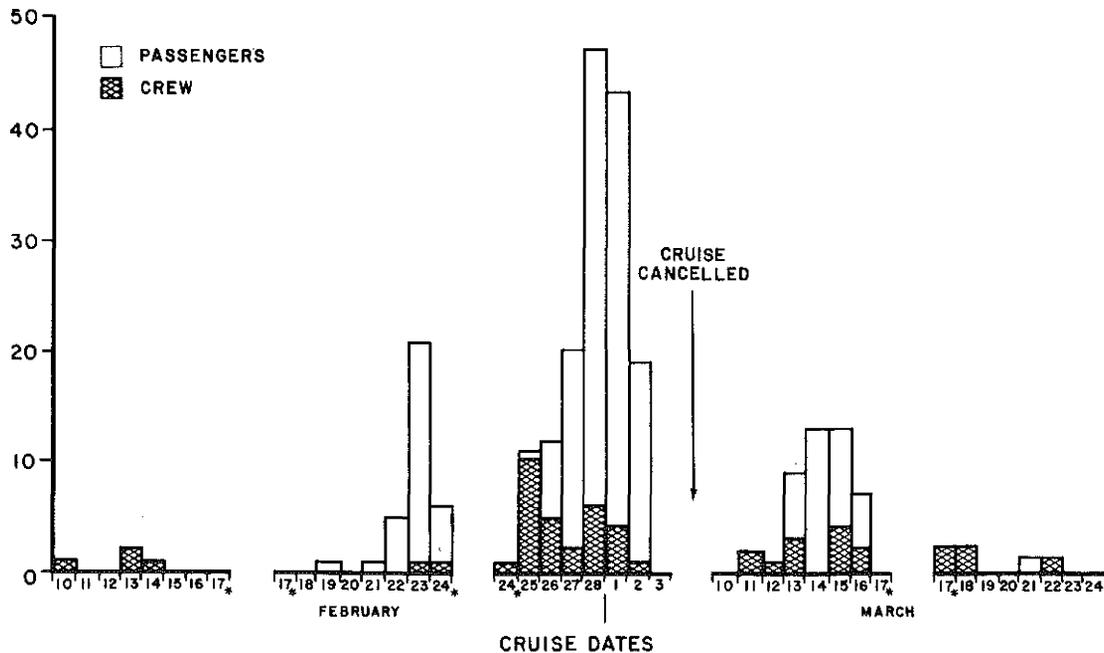
A line listing of outbreaks of unknown etiology may be obtained by writing to the Enteric Diseases Branch, Bacterial Diseases Division, Center for Infectious Diseases, Centers for Disease Control, Atlanta, Georgia 30333.

I. Selected Foodborne Outbreak Articles, 1979, Taken From Morbidity and Mortality Weekly Report

Salmonella heidelberg Gastroenteritis Aboard a Cruise Ship
(MMWR 1979;28(13):145-7)

An outbreak of gastrointestinal illness occurred aboard the T.S.S. Festivale, a Caribbean cruise ship of Panamanian registry owned and operated by Carnival Cruise Lines, on its February 17-24, 1979, cruise. The outbreak was detected when several passengers who were ill aboard ship notified the Dade County Health Department and the U.S. Quarantine Office after they disembarked in Miami. On the evening of February 26, a quarantine officer in San Juan, where the ship was docked, reviewed the ship's medical log and noted that the outbreak had begun on February 22 and that 32 (3%) of the 1,149 passengers had been seen by the physician for a diarrheal illness during the cruise (Figure 1). An outbreak was also apparently occurring on the February 24-March 3 cruise: by February 26, 26 (2%) of the 1,160 passengers and 18 (3%) of the 540 crew had reported having diarrhea to the ship's physician and many more passengers were complaining of a gastrointestinal illness. A Public Health Service (PHS) quarantine officer and a PHS sanitarian boarded the ship in St. Martin on February 28 to begin an epidemiologic and environmental investigation.

Fig.1 CLINIC VISITS FOR DIARRHEAL ILLNESS AMONG PASSENGERS AND CREW ON 4 CRUISES OF THE T. S. S. FESTIVALE, FEBRUARY 10 - MARCH 24, 1979



* HALF DAY

A questionnaire survey was conducted on March 1. Of the 1,129 (97%) passengers responding, 379 (34%) reported a gastrointestinal illness defined as either watery diarrhea or severe cramps and vomiting; 108 passengers became ill within 48 hours of boarding the ship on February 24. Stool cultures previously obtained from 4 passengers ill during an earlier cruise and from 14 ill crew members, removed from the ship when it docked in St. Thomas on February 27, grew Salmonella group B.

A sanitation inspector for the Quarantine Division inspected the ship on March 2. The water was found to have adequate levels of residual chlorine and to be negative for coliforms. However, multiple deficiencies in sanitation were found, particularly in food handling and preparation. Records revealed that the ship had not passed earlier sanitation inspections conducted by the Quarantine Division.

On March 3 a second questionnaire was distributed concerning food consumed during the cruise of February 24-March 3. The survey, completed by 93% of passengers, implicated

turkey and macaroni salad served at the evening buffet on February 24 as vehicles of transmission. Stool cultures were obtained from 21 ill passengers and 6 well passengers before the ship docked; S. heidelberg was isolated from 17 (81%) of the ill and 4 (67%) of the well passengers. The same Salmonella serotype was cultured from 7 of 35 different food specimens taken from the ship's galley on March 1 and 2. However, the original turkey and macaroni salad from the evening buffet of February 24 were no longer available. Stool specimens were obtained from 269 food handlers and tested for salmonellae, and through April 6 more than 60 had been positive for Salmonella group B. The food handlers were employees of Apollo caterers, a Miami-based firm that caters meals on cruise ships.

The following recommendations were made: 1) remove and destroy leftover foods, 2) completely clean and sanitize the galley, 3) screen food handlers for Salmonella and remove all those who are positive, 4) make structural improvements in the kitchen's refrigeration systems and dishwashing areas, and 5) provide better supervision and education of galley crew to improve food handling practices. Since these changes would take at least 1 week to implement, the PHS recommended that the company cancel the March 3-10 cruise. The company agreed to cancel the cruise and implement the recommendations.

On March 10, the T.S.S. Festivale sailed again with a large number of new galley crew members replacing those who had positive Salmonella cultures. A small outbreak of gastrointestinal illness occurred during this cruise (Figure 1), and S. heidelberg was isolated from 1 new passenger. During the subsequent cruise, which began March 17, only 1 of more than 1,100 passengers reported to the ship's doctor with diarrhea.

Editorial Note: While shipboard outbreaks of gastrointestinal illness occur yearly (1-2), this is the first time since 1973 that CDC has recommended that a cruise be canceled because of an outbreak (3). The epidemiologic data and the isolation of S. heidelberg from food handlers and food specimens suggested that the ship's principal problems were in the preparation and storage of food.

According to quarantine regulations, the master of a vessel is required to report to the Quarantine Station, within 24 hours before arriving in port, the number of passengers and crew who were seen by the ship's physician for the treatment of diarrhea. CDC usually conducts an epidemiologic and environmental investigation when 3% or more of passengers and crew members experience a diarrheal illness.

The Quarantine Division routinely inspects and scores cruise ships for their adherence to sanitation codes. The results of sanitation inspections on individual cruise ships as well as a monthly summary of the results of the most recent inspections of all cruise ships sailing from or calling at a U.S. port may be obtained from the U.S. Public Health Service, 1015 North American Way, Room 107, Miami, Florida 33132.

References

1. Merson MH, Hughes JM, Lawrence DN, Wells JG, D'Agnese JJ, Yashuk JC. Food and waterborne disease outbreaks on passenger cruise vessels and aircraft. *Journal of Milk and Food Technology* 1976;39:285-8.
2. Merson MH, Hughes JM, Wood BT, Yashuk JC, Wells JG. Gastrointestinal illness on passenger cruise ships. *JAMA* 1975;231:723-7.
3. *MMWR* 1973;22:217-8.

Staphylococcal Food Poisoning Associated with Genoa and Hard Salami--United States (*MMWR* 1979;28(15):179-80)

Since January 1, 1979, 8 incidents of staphylococcal food poisoning associated with salami products produced by the Patrick Cudahy, Inc. plant, Establishment 28, Cudahy, Wisconsin, have been reported. The reports came from Pennsylvania (4), Virginia (2), Minnesota (1), and Wisconsin (1). Nineteen persons have become ill with symptoms compatible with staphyloenterotoxigenosis after an average incubation period of 4 hours. At least 7 persons were hospitalized.

Although laboratory analysis of remaining specimens of the implicated salami did not reveal Staphylococcus enterotoxin or high counts of S. aureus, investigators found that the procedure used by the company to manufacture the salami did not provide adequate controls to prevent staphylococcal growth and concomitant enterotoxin production. In addition, analysis of other products with the same establishment code and lot numbers as the salami associated

with illness revealed counts of coagulase-positive staphylococci ranging from 16,000 to 930,000 organisms per gram; Staphylococcus enterotoxin was identified in 1 lot.

On March 9, the U.S. Department of Agriculture (USDA) announced a voluntary recall of 4 implicated lots of 4 oz., sliced, vacuum-packaged Genoa salami with labels marked "sell by" 1 of 4 dates: February 25, March 9, March 30, and April 20. Since that announcement, 4 more outbreaks have occurred associated with products not involved in the initial recall; Genoa and hard salamis, sliced to order from whole sticks sold in groceries and delicatessens, were implicated. Analysis of random sticks of these 2 types of salami from Establishment 28, found in marketing channels, revealed counts of coagulase-positive staphylococci ranging from 0 to $>10^6$ organisms/g. Independent laboratory testing of company-supplied samples of Genoa salami, obtained by USDA at Establishment 28 after the recall revealed counts of coagulase-positive staphylococci ranging from 2,600 to $>10^6$ organisms/g. One specimen also contained Staphylococcus enterotoxin C. On April 13, on the basis of these findings, the manufacturer voluntarily recalled its Genoa salami and hard salami produced at Establishment 28.

Editorial Note: In the production of fermented sausage, lightly salted meat is intentionally temperature-controlled to allow lactobacilli to grow; these usually inhibit the growth of other organisms. However, if the procedure is not adequately monitored, S. aureus organisms may multiply on the surface of the sausage and produce enterotoxin. The typical 1- to 2-month curing period for sausage will eventually cause these Staphylococcus organisms to die off, but the enterotoxin--which causes human illness--will remain. Detection of enterotoxin is difficult because (1) it is found only in the outer, one-eighth inch surface of the salami and then only in random locations (it varies from salami to salami and within individual sticks); and (2) the in vitro tests used to detect its presence are not sufficiently sensitive to detect small amounts.

Staphylococcal Food Poisoning--Delaware (MMWR 1979;28(37):445-6)

On March 10, 1979, 64 cases of acute gastrointestinal disease occurred among 107 guests at a wedding reception in Sussex County, Delaware.

Symptoms included vomiting (85%), nausea (74%), abdominal cramps (61%), and diarrhea (39%). Thirty-eight of those affected sought emergency room attention, although none were hospitalized. Incubation period of the illness ranged from 1.6 to 6.5 hours, with a median of 3.5 hours.

Food histories, obtained from 103 of the guests, implicated chicken salad as the food associated with illness. The attack rate among those who ate chicken salad was 76% (62/82), while only 9% (2/21) of those who did not eat the salad became ill ($p<.001$). Coagulase-positive Staphylococcus aureus was subsequently isolated from the chicken salad and the food grinder used to prepare it. No skin lesions were evident on any of the 6 food handlers, but S. aureus was cultured from nasal swabs of 3. Phage typing, performed at CDC, demonstrated that the isolates from the chicken salad, the food grinder, and the nasal swab from the person who prepared chicken salad were all type 95.

The food was mostly prepared in private homes. The chicken for the salad was cooked and deboned on March 8 and refrigerated in a large plastic washtub. The following day the chicken was ground in a meat grinder with celery and onions, mixed with mayonnaise, and then refrigerated in the same tub. On the day of the reception, the salad was not refrigerated during transport or before or during the reception--a period of approximately 7 hours. During serving, it was noted that the chicken salad from the central portion of the container felt warmer than that from the top, indicating uneven refrigeration.

Editorial Note: This classic staphylococcal outbreak underscores the need for continuing public education in proper food handling, particularly with regard to prompt and adequate refrigeration of prepared foods. Staphylococcal food poisoning has been recognized since 1914, when an outbreak in the Philippines, caused by inadequate refrigeration of milk from a cow with a chronic staphylococcal infection, was described (1). This type of food poisoning remains a major cause of outbreaks of acute gastrointestinal disease, constituting approximately 25% of all foodborne outbreaks of known etiology reported to CDC between 1972 and 1977.

The illness is caused by the presence of a heat-stable enterotoxin produced by only a few strains of S. aureus, often from phage group 3; phage typing alone, however, cannot determine whether a given strain will produce enterotoxin.

The vehicle of transmission in staphylococcal food poisoning is almost always a protein-containing food. Ham is the most common vehicle in the United States, where it is implicated in 28% of outbreaks. Contamination, as in this case, is usually assumed to be from food handlers; use of improper holding temperatures allows multiplication of the staphylococci and elaboration of the toxin. After ingestion, the incubation period may range from 30 minutes to 8 hours, with vomiting the predominant symptom. The illness produced may be quite severe, although short-lived; a few fatal cases have been reported (2).

Bacillus cereus may cause a similar clinical syndrome mediated by a heat-stable emetic toxin; the median incubation period is less than 6 hours, with illness characterized by vomiting and abdominal cramps (3). B. cereus is also capable of producing a heat-labile diarrheal toxin, which may mimic Clostridium perfringens (4).

References

1. Barber MA. Milk poisoning due to a type of Staphylococcus albus occurring in the udder of a healthy cow. Philippine Journal of Science 1914;98:515-9.
2. Currier RW, Taylor A, Wolf FS, Warr M. Fatal staphylococcal food poisoning. South Med J 1973;66:703-5.
3. Terranova W, Blake PA. Bacillus cereus food poisoning. N Engl J Med 1978;298:143-4.
4. Turnbull PCB, Kramer JM, Jorgensen K, Gilbert RJ, Melling J. Properties and production characteristics of vomiting, diarrheal, and necrotizing toxins of Bacillus cereus. Am J Clin Nutr 1979;32:219-28.

Shigellosis in a Children's Hospital--Pennsylvania (MMWR 1979;28(42):498-9)

An outbreak of shigellosis occurred May 17-30, 1979, among hospital employees in a children's hospital in Pennsylvania. Thirty-two percent of employees reported being ill; 280 employees and visitors with complaints of vomiting and/or diarrhea presented to the employee health service and were cultured; 142 (51%) had positive stool cultures for Shigella sonnei. Staffing problems during the outbreak were severe, and the hospital was closed to new admissions for a 3-day period.

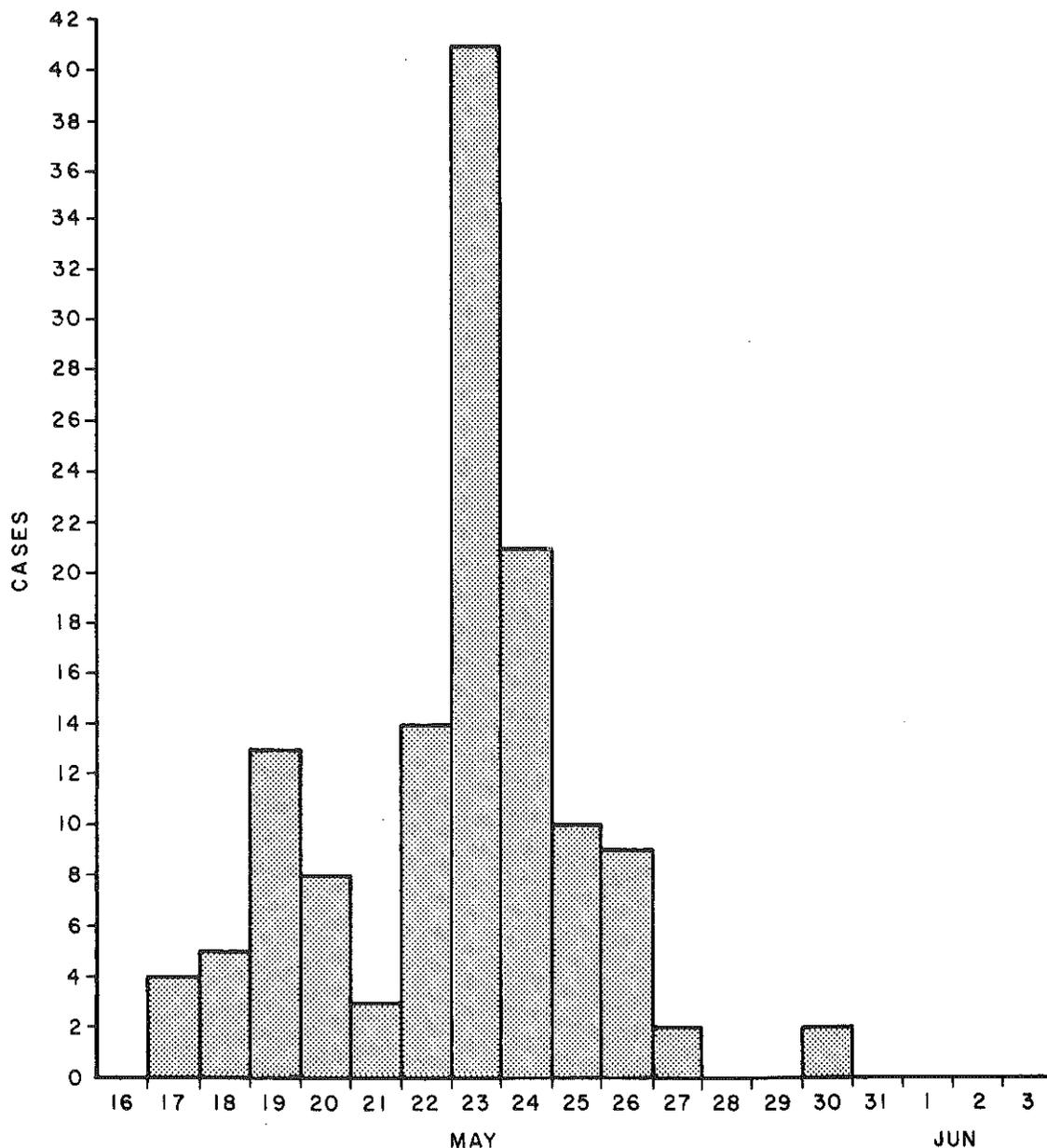
Questionnaires were sent to 1,700 employees to determine the symptoms of disease and places where these persons had eaten from May 16-21; a food-specific history was obtained from those who had eaten in the hospital cafeteria. One thousand ninety-three questionnaires (64%) were returned. Analysis showed a strong association between illness and eating in the hospital cafeteria ($p < .0001$). Based on 78 culture-confirmed cases and 150 well controls, significant associations were found between illness and consumption of tuna salad ($p < .0001$) and eating food from the salad bar ($p < .0001$). No association between illness and consumption of hot foods was found.

One cafeteria employee had diarrhea on May 17, the first day of the outbreak. She had been exposed to a child with severe diarrhea at home before onset of her illness. This employee was found to be culture-positive for S. sonnei. She had worked on May 17 and May 21 and was responsible for preparing all salads and sandwiches in the employee cafeteria, where visitors also ate sometimes. The 2 peaks in the outbreak were on May 19 and May 23--consistent with the 1- to 2-day incubation period of foodborne shigellosis (Figure 1).

The organism identified from culture-positive individuals was resistant to ampicillin and tetracycline and sensitive to trimethoprim-sulfamethoxazole. All symptomatic individuals were treated with a 5-day course of the latter drug, or with furazolidone, if they were sensitive to sulfa. For cafeteria employees, 3 negative rectal cultures--taken at 1-day intervals at least 48 hours after antibiotic therapy had ended--were required before a culture-positive individual could return to work. Other culture-positive hospital employees were permitted to return to work after 48 hours of therapy. No hospitalized patients became culture positive for Shigella as a result of the outbreak.

Editorial Note: Shigella organisms remain a major cause of gastrointestinal illness in the United States: 15,336 isolates were reported to CDC in 1978 (1). Although transmission is usually from person to person, in the 18-year period from 1961 through 1978 there were 84 reported outbreaks of common-source foodborne illness due to Shigella. Unlike most Salmonella

Fig. 1 INDIVIDUALS CULTURE-POSITIVE FOR *SHIGELLA*, BY DATE OF ONSET, A CHILDREN'S HOSPITAL, PENNSYLVANIA, MAY 1979*



*EXCLUDES CASES IN WHICH DATE OF ONSET WAS UNKNOWN

species, *Shigella* are host specific for man and generally survive poorly in the environment. When foodborne outbreaks do occur, they can almost always be traced to contamination of food by an infected food handler. As in this case, the vehicle in foodborne *Shigella* outbreaks is typically a salad or other food in which the preparation requires extensive handling of ingredients. Foodborne *Shigella* outbreaks are frequently large and have a high attack rate. For foodborne *Shigella* outbreaks from 1961 to 1975, the average attack rate was 47%, with an average outbreak size of 148 persons (2).

The procedures used in this instance to evaluate food-service employees before their return to work followed the recommendations of the American Public Health Association—i.e., that cultures be obtained 48 hours after cessation of therapy and that they be obtained at least 24 hours apart (3). More specific regulations relating to food-service employees—such

as the number of cultures, the amount of time that should elapse between ceasing therapy and starting post-therapy culturing, and the time between cultures--vary from state to state, and there is no single combination of these variables which has been shown to be clearly superior in identifying infectious individuals.

No secondary spread from members of the hospital staff to patients occurred in this outbreak. This contrasts with studies in households, in which up to 35% of children present in the household have been shown to become infected with Shigella after an initial infection in 1 adult household member (4).

References

1. MMWR 1979;28:486-7.
2. Black RE, Craun GF, Blake PA. Epidemiology of common-source outbreaks of shigellosis in the United States, 1961-1975. Am J Epidemiol 1978;108:47-52.
3. Benenson AS (ed). Control of Communicable Diseases in Man. Washington, American Public Health Assoc, 1975.
4. Thomas MEM, Tillett HE. Dysentery in general practice: A study of cases and their contacts in Enfield and an epidemiological comparison with salmonellosis. J Hyg (Camb) 1973;71:373-89.

Non-01 Vibrio cholerae Infections--Florida (MMWR 1979;28(48):571-577)

Since November 8, 1979, non-01 Vibrio cholerae organisms have been isolated from the stools of 3 persons who presented to a single hospital in northern Florida. Raw oysters harvested from or near Oyster Bay, Wakulla County, Florida, have been epidemiologically incriminated as the vehicle of transmission.

The first patient, a 24-year-old woman, became ill with nausea, vomiting, abdominal cramps, and bloody diarrhea on November 8, 30 hours after consuming raw oysters harvested at Mashles Sand near Oyster Bay. She was admitted to the hospital on November 9, was treated with intravenous fluids, and recovered.

The second patient was a 25-year-old man who developed watery diarrhea, vomiting, and abdominal cramps on November 12, 15 hours after he had eaten raw oysters harvested at Purify Creek on Oyster Bay. He was seen in the hospital emergency room, but he was not clinically dehydrated and was discharged after receiving symptomatic therapy.

The third patient, a 23-year-old man, became ill with nausea, vomiting, abdominal cramps, and bloody diarrhea on November 18, 12 hours after he had consumed raw oysters obtained from a supplier in Wakulla County. These oysters were thought to have been harvested from Oyster Bay. He was mildly dehydrated, was admitted to the hospital on November 18, and was discharged after 24 hours of intravenous fluid therapy.

The raw oysters were consumed by these 3 patients at family and social gatherings. Another 8 persons were identified who had onset of diarrheal illness within 48 hours after they had eaten raw oysters at these occasions.

Investigation of 11 adult control patients with diarrhea, admitted to the same hospital during November 8-24, but with stool cultures negative for V. cholerae non-01, revealed that none had consumed raw oysters within 48 hours before admission ($p < .01$). Water and oyster samples collected from the areas where oysters were harvested by the first 2 patients had elevated fecal coliform counts. These areas have been temporarily closed to oyster harvesting by state regulatory authorities, and the open and closed areas in and around Oyster Bay are being monitored for fecal coliform bacteria twice a week.

Editorial Note: The species V. cholerae now includes not only the strains that cause cholera epidemics (V. cholerae O group 1) but also organisms that are similar biochemically and by DNA homology to the epidemic strains but which have not been associated with epidemic disease (V. cholerae of other O groups, or non-01 V. cholerae). The latter were formerly referred to as non-agglutinating vibrios (NAGs) or non-cholera vibrios (NCVs).

Sporadic cases of disease associated with isolation of non-01 V. cholerae do occur in the United States (1). Although some of these cases have been anecdotally associated with eating raw shellfish, in this instance raw oysters were epidemiologically incriminated. In the first 2 cases reported here, the incriminated oysters came from areas with elevated fecal coliform counts, suggesting that there was fecal contamination of the areas. Consumption of raw shellfish from contaminated waters carries a significant health risk. Other diseases,

including hepatitis and viral gastroenteritis, have occurred after consumption of contaminated shellfish (2). In Florida and other states, regulatory authorities monitor, under the National Shellfish Sanitation Program, the fecal coliform counts of oyster beds harvested for commercial distribution. At the federal level, this program is administered by the U.S. Food and Drug Administration.

References

1. Hughes JM, Hollis DG, Gangarosa EJ, Weaver RE. Non-cholera vibrio infections in the United States: Clinical, epidemiologic and laboratory features. *Ann Intern Med* 1978;88:602-6.
2. Earampamoorthy S, Koff RS. Health hazards of bivalve-mollusk ingestion. *Ann Intern Med* 1975;83:107-10.

Viral Hepatitis Outbreaks--Georgia, Alabama (MMWR 1979;28(49):581)

Ten recent cases of probable hepatitis A associated with consumption of raw oysters from Florida have been identified in Albany, Georgia, and Mobile, Alabama.

An investigation of 3 Albany residents in whom hepatitis was diagnosed during the week of October 28 disclosed that 2 had eaten raw oysters on October 13, and the other had eaten raw oysters on October 15. The oysters had all come from a single sack purchased in Florida.

An investigation of 5 Mobile residents with onset of hepatitis in the period November 5-7 found that their only common exposure was having eaten raw oysters at a club dinner on October 11. Two other Mobile patients with hepatitis who had eaten raw oysters purchased from the same store at the same time as the oysters purchased to serve at the club dinner, were also identified.

The Food and Drug Administration, CDC, and state and local health authorities are trying to trace the source of the oysters for both outbreaks. Preliminary results suggest that the oysters came from a single area in Florida. The investigation is continuing.

Editorial Note: Raw oysters have been implicated as the vehicle of transmission for hepatitis in several outbreaks in the United States, most recently in 1973, when 285 people became ill after eating raw oysters harvested in Louisiana (1). The number of cases involved in the 2 outbreaks reported here is small compared with previous outbreaks, although there may be additional cases which have not yet been identified. Physicians are urged to report all cases of hepatitis to the appropriate public health authorities and to be particularly alert to possible oyster-associated cases.

Reference

1. Portnoy BL, Mackowiak PA, Caraway CT, Walker JA, McKinley TW, Klein CA. Oyster-associated hepatitis. Failure of shellfish certification programs to prevent outbreaks. *JAMA* 1975;233:1065-8.

Follow-up on Viral Hepatitis Outbreaks--Alabama, Georgia (MMWR 1979;28(50):594-5)

The origin of oysters associated with 7 cases of hepatitis in Mobile, Alabama, and 3 cases of hepatitis in Albany, Georgia (1), has been traced to Apalachicola Bay, Florida.

By obtaining descriptions of oyster packaging and studying invoices of oyster dealers, the investigators traced shucked oysters consumed in the Mobile hepatitis outbreak to dealers that handled oysters harvested exclusively from Apalachicola Bay. Oysters associated with the Georgia outbreak had been purchased as shell stock from a different dealer, who also used Apalachicola Bay oysters exclusively. In neither investigation were any persons who were involved in the harvesting or handling of the oysters before their consumption identified as having hepatitis. The exact growing area of the incriminated oysters in Apalachicola Bay were not identified. The most probable dates of harvesting of the incriminated oysters were September 25-26 for the Georgia cases and October 6-8 for the Alabama cases. No cases of hepatitis related to the consumption of raw oysters from Apalachicola Bay have been identified with dates of onset after November 8.

During the last week of September and first week of October, fecal coliform counts transiently exceeded the recommended standard of 14 coliforms* per 100 ml of water (2) at several stations of the bay that were open for oyster harvesting. These counts ranged from 23 to 240 coliforms MPN* with a median of 49. Because of these high coliform counts, 1 area of the bay was subsequently closed to oyster harvesting October 4 by the Florida Department of Natural Resources.

Editorial Note: This investigation illustrates the problems of identifying the precise cause of contamination of shellfish so that preventive measures can be taken. It was difficult to trace the oysters to Apalachicola Bay and impossible to locate the exact growing area in the bay since Florida does not require labeling of oysters to indicate their place of harvesting.

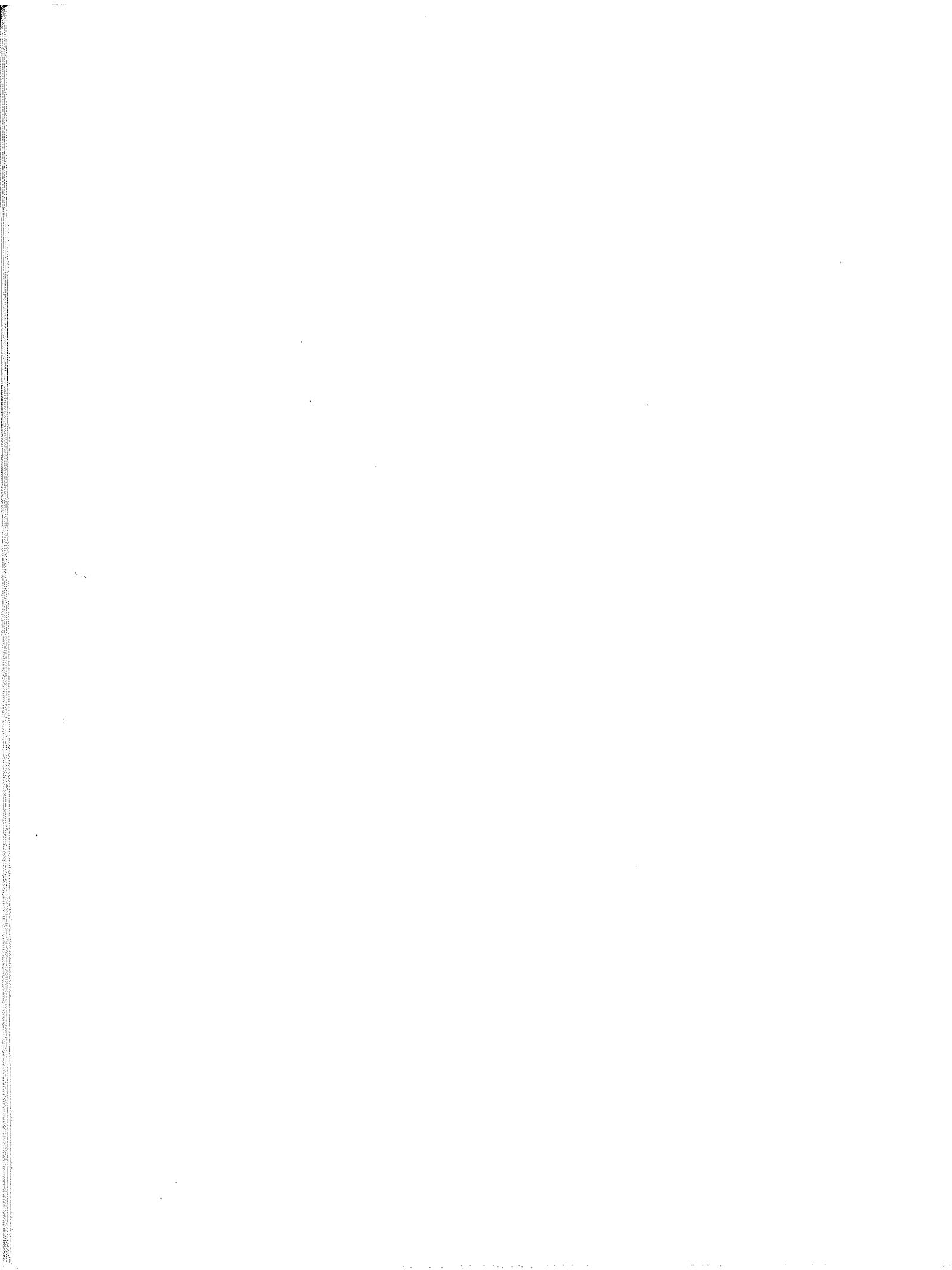
Several hypotheses can be advanced to explain transient contamination of oyster beds in the bay: increased run-off associated with heavy rains caused by hurricane Frederick during mid-September, illegal dumping of sewage from passing boats, and illegal disposal of waste from land sources. Since large numbers of oysters are harvested from the bay, and only a few cases of oyster-associated hepatitis have been recognized, it seems likely that only a small proportion of oysters from the bay harbored hepatitis virus. The apparent lack of new cases suggests that the problem may have abated.

In addition to hepatitis, fecally contaminated shellfish have been associated with outbreaks of typhoid fever, cholera, and viral (Norwalk agent) gastroenteritis (3-5). In all these outbreaks the shellfish were eaten raw or undercooked. Well-cooked shellfish do not appear to be associated with a risk of acquiring hepatitis. Strict enforcement and scrupulous compliance with all shellfish sanitation regulations should minimize the risk of disease caused by fecally contaminated shellfish.

References

1. Centers for Disease Control. Viral Hepatitis Outbreaks--Georgia, Alabama. Morbidity Mortality Weekly Rep 1979;28:581.
2. Food and Drug Administration. National Shellfish Safety Program: Proposed rulemaking. Federal Register 1975;40:25930.
3. Lumsden LL, Hasseltine HE, Leake JP, et al. A typhoid-fever epidemic caused by oyster-borne infection (1924-1925). Public Health Rep 1975;50(suppl):1-102.
4. Blake PA, Rosenberg ML, Bandeira Costa J, et al. Cholera in Portugal, 1974. 1. Modes of transmission. Am J Epidemiol 1977;105:337-43.
5. Murphy AM, Grohmann GS, Christopher PJ, et al. An Australia-wide outbreak of gastroenteritis from oysters caused by Norwalk virus. Med J Aust 1979;2:329-33.

*Most probable number (MPN) index.



J. Bibliography

GENERAL

1. Bryan FL. Emerging foodborne diseases. I. Their surveillance and epidemiology. II. Factors that contribute to outbreaks and their control. *J Milk Food Technol* 1972;35:618-25, 632-8.
2. Bryan FA. Factors that contribute to outbreaks of foodborne disease. *J of Food Protection* 1978;41:816-27.
3. Bryan FL. Foodborne diseases in the United States associated with meat and poultry. *J of Food Protection* 1980;43:140-50.
4. Food Research Institute. Annual report 1979, University of Wisconsin, Madison, Wisconsin.
5. Horwitz MA. Specific diagnosis of foodborne disease. *Gastroent* 1977;73:375-81.
6. Riemann H, ed. Foodborne infections and intoxications. New York: Academic Press, 1969.
7. Sours HE, Smith DG. Outbreaks of foodborne disease in the United States, 1972-1978. *J Infect Dis* 1980;142:122-5.

BACTERIAL

Bacillus cereus

1. Giannella RA, Brasile L. A hospital foodborne outbreak of diarrhea caused by Bacillus cereus: Clinical, epidemiologic, and microbiologic studies. *J Infect Dis* 1979;139:366-70.
2. Mortimer PR, McCann G. Food poisoning episodes associated with Bacillus cereus in fried rice. *Lancet* 1974;1:1043-5.
3. Terranova W, Blake PA. Bacillus cereus food poisoning. *N Engl J Med* 1978;298:143-4.
4. Turnbull PCB, Kramer JM, Torgensen K, Gilbert RJ, Melling J. Properties and production characteristics of vomiting, diarrheal, and necrotizing toxins of Bacillus cereus. *Am J Clin Nutr* 1979;32:219-28.

Brucella

1. Buchanan TM, et al. Brucellosis in the United States 1960-1972. *Med* 1974;53:403-39.
2. Fox MD, Kaufman AF. Brucellosis in the United States, 1965-1974. *J Infect Dis* 1977;136:312-6.
3. Spink WW. The nature of brucellosis. Minneapolis, Lund Press, Inc., 1956.
4. Young EJ, Suvannaparrat U. Brucellosis outbreak attributed to ingestion of unpasteurized goat cheese. *Arch Intern Med* 1975;135:240-3.

Campylobacter

1. Blaser MJ, Craven J, Powers BW, LaForce FM, Wang W-LL. Campylobacter enteritis associated with unpasteurized milk. *Am J Med* 1979;67:715-8.

Clostridium botulinum

1. Center for Disease Control. Botulism in the United States, 1899-1977. Handbook for Epidemiologists, Clinicians, and Laboratory Workers, CDC, Atlanta, May 1979.
2. Cherington M. Botulism. Ten-year experience. *Arch Neurol* 1974;30:432-7.
3. Dowell VR Jr, McCroskey LM, Hatheway CL, et al. Coproexamination for botulinal toxin and Clostridium botulinum - A new procedure for laboratory diagnosis of botulism. *JAMA* 1977;238:1829-32.
4. Koenig MG, Spichard A, Cardella MA, et al. Clinical and laboratory observations of type E botulism in man. *Med* 1964;43:517-45.
5. Koenig MG, Drutz DJ, Mushlin AI, et al. Type B botulism in man. *Am J Med* 1967;42:208-19.
6. Morris JG, Hatheway CL. Botulism in the United States, 1979. *J Infect Dis* 1980;142:302-5.

Clostridium perfringens

1. Bryan FL. What the sanitarian should know about Clostridium foodborne illness. *J Milk Food Technol* 1969;32:381-9.

2. Lowenstein MS. Epidemiology of Clostridium perfringens food poisoning. N Engl J Med 1972;286(19):1026-7.

3. Stringer MF, Turnbull PCB, Gilbert RJ. Application of serological typing to the investigation of outbreaks of Clostridium perfringens food poisoning, 1970-1978. J Hyg (Camb) 1980;84:443-56.

Escherichia coli

1. Marier R, Wells JG, Swanson RC, Callahan W, Mehlman IJ. An outbreak of enteropathogenic Escherichia coli foodborne disease traced to imported French cheese. Lancet 1973;2:1376-8.

2. Sack RB. Human diarrheal disease caused by enterotoxigenic Escherichia coli. Ann Rev Microbiol 1975;29:333-53.

Salmonella

1. Aserkoff B, Schroeder SA, Brachman PS. Salmonellosis in the United States--A five-year review. Am J Epidemiol 1970;92:13-24.

2. Bryan FL. What the sanitarian should know about salmonellae and staphylococci in non-dairy foods. II. Salmonellae. J Milk Food Technol 1968;31:131-40.

3. Cohen ML, Blake PA. Trends in foodborne salmonellosis outbreaks: 1963-1975. J Food Protection 1977;40:798-800.

Shigella

1. Black RE, Craun GF, Blake PA. Epidemiology of common-source outbreaks of shigellosis in the United States, 1961-1975. Am J Epidemiol 1978;108:47-52.

2. Donadio JA, Gangarosa EJ. Foodborne shigellosis. J Infect Dis 1969;119:666-8.

Staphylococcus

1. Bryan FL. What the sanitarian should know about salmonellae and staphylococci in non-dairy foods. I. Staphylococci. J Milk Food Technol 1968;31:110-16.

2. Merson MH. The epidemiology of staphylococcal foodborne disease. Proceedings of Staphylococci in Foods Conference, Pennsylvania State University, University Park, Pennsylvania, 1973, pp 20-37.

3. Minor TE, Marth EH. Staphylococcus aureus and staphylococcal food poisoning. J Milk Food Technol 1972;34:21-39, 77-83, 227-241, 1973;35:447-76.

Group A Streptococcus

1. Hill HR, Zimmerman RA, Reid GVK, Wilson E, Kitton RM. Foodborne epidemic of streptococcal pharyngitis at the United States Air Force Academy. N Engl J Med 1969;280:917-21.

2. McCormick JB, Kay D, Hayes M, Feldman RA. Epidemic streptococcal sore throat following a community picnic. JAMA 1976;236:1039-41.

Vibrio cholerae O1

1. Blake PA, Allegra DT, Snyder JD, Barrett TJ, McFarland L, Caraway CT, Feeley JC, Craig JP, Lee JV, Puhr ND, Feldman RA. Cholera--A possible endemic focus in the United States. N Engl J Med 1980;302:305-309.

2. Gangarosa EJ, Mosley WH. Epidemiology and surveillance of cholera. Cholera, edited by Barua D, Burrows W. Philadelphia, London, Toronto, WB Saunders Co., 1974, p 381.

Vibrio cholerae Non-O1

1. Hughes JM, Hollis DG, Gangarosa EJ, Weaver RE. Non-cholera vibrio infections in the United States--Clinical, epidemiologic, and laboratory features. Ann Intern Med 1978;88:602-6.

Vibrio parahaemolyticus

1. Barker WH. Vibrio parahaemolyticus outbreaks in the United States. Lancet 1974;1:551-4.

2. Barrow GI, Miller DC. Vibrio parahaemolyticus and seafoods. Soc Appl Bacteriol Symp Ser 1976;4:181-95.

3. Dadisman TA Jr, Nelson R, Molenda JR, et al. Vibrio parahaemolyticus gastroenteritis in Maryland. I. Clinical and epidemiologic aspects. Am J Epidemiol 1972;96:414-26.

4. International Symposium on Vibrio parahaemolyticus, September 17-18, 1973, Fujino, Sakaguchi G, Sakazaki R, Takeda (ed). Saikon Publishing Co., Ltd., Tokyo, Japan, 1974.

CHEMICAL

Heavy Metal

Cadmium

1. Baker TD, Hafnew WG. Cadmium poisoning from a refrigerator shelf used as an improvised barbecue grill. Public Health Rep 1961;76:543-4.

Copper

1. Hopper SH, Adams HS. Copper poisoning from vending machines. Public Health Rep 1958;73:910-4.

2. Semple AB, Parry WH, Phillips DE. Acute copper poisoning: An outbreak traced to contaminated water from a corroded geyser. Lancet 1960;2:700-1.

Tin

1. Barker WH, Runte V. Tomato juice-associated gastroenteritis. Washington and Oregon, 1969. Am J Epidemiol 1972;96:219-26.

Zinc

1. Brown MA, Thom JV, Orth GL, et al. Food poisoning involving zinc contamination. Arch Environ Health 1964;8:657-60.

Ciguatera

1. Bagnis R, Chanteau S, Chungue E, Huntel JM, Yasumoto T, Inoue A. Origins of ciguatera fish poisoning: A new dinoflagellate, Gambierdiscus toxicus Adachi and Fukuyo, definitely involved as a causal agent. Toxicon 1980;18:199-208.

2. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 325-402.

3. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.

4. Lawrence DN, Enriquez MB, Lumish RM, Maceo A. Ciguatera fish poisoning in Miami. JAMA 1980;244:254-8.

Puffer Fish (tetrodotoxin)

1. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 437-548.

2. Torda TA, Sinclair E, Ulyatt DB. Puffer fish (tetrodotoxin) poisoning: Clinical record and suggested management. Med J Aust 1973;1:599-602.

Scombrototoxin

1. Arnold SH, Brown WD. Histamine toxicity from fish products. Advances in Food Research 1978;24:113-54.

2. Gilbert RJ, Hobbs G, Murray CK, Cruickshank JG, Young SEJ. Scombrototoxic fish poisoning: Features of the first 50 incidents to be reported in Britain (1976-1979). British Med J 1980;281:71-2.

3. Halstead BW. Poisonous and venomous marine animals of the world. Princeton, The Darwin Press, 1978, pp 417-35.

4. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.

5. Merson MH, Baine WB, Gangarosa EJ, Swanson RC. Scombroid fish poisoning: Outbreak traced to commercially canned tuna fish. JAMA 1974;228:1268-9.

Monosodium Glutamate

1. Schaumburg HH, Byck R, Gerstl R, Mashman JH. Monosodium L-glutamate: its pharmacology and role in the Chinese restaurant syndrome. Science 1969;163:826-8.

Mushroom Poison

1. Becker CE, et al. Diagnosis and treatment of Amanita phalloides type mushroom poisoning - use of thiotic acid. West J Med 1976;125:100-9.

2. Benedict RG. Mushroom toxins other than Amanita, Kadis S, Ciegler A, Aji SJ: Microbial toxins, Vol 8 Fungal toxins, New York and London, Academic Press, 1972, pp 281-320.

3. Mitchel DH. Amanita mushroom poisoning. Ann Rev Med 1980;31:51-7.

4. Tyler VE. Poisonous mushrooms: Progress in chemical toxicology. Vol 1, edited by Stolman A, New York Academic Press, 1963, pp 339-84.

Paralytic and Neurotoxic Shellfish Poison

1. Halstead BW. Poisonous and venomous marine animals of the world. The Darwin Press, Princeton, 1978, pp 43-78.

2. Hughes JM, Merson MH. Fish and shellfish poisoning. N Engl J Med 1976;295:1117-20.
3. Music SI, Howell JT, Brumback CL. Red tide: its public health implications. J Fla Med Assoc 1973;60:27-9.

PARASITIC

Anisakidae

1. Chitwood MD. Nematodes of medical significance found in market fish. Am J Trop Med Hyg 1970;19:599-602.

T. spiralis

1. Gould SE. Trichinosis in man and animals. Springfield, Ill, Charles C. Thomas, 1970.
2. Zimmerman WJ, Steele JH, Kagan IG. Trichinosis in the U.S. population 1966-1970--Prevalence and epidemiologic factors. Health Services Rep 1973;88:606-23.

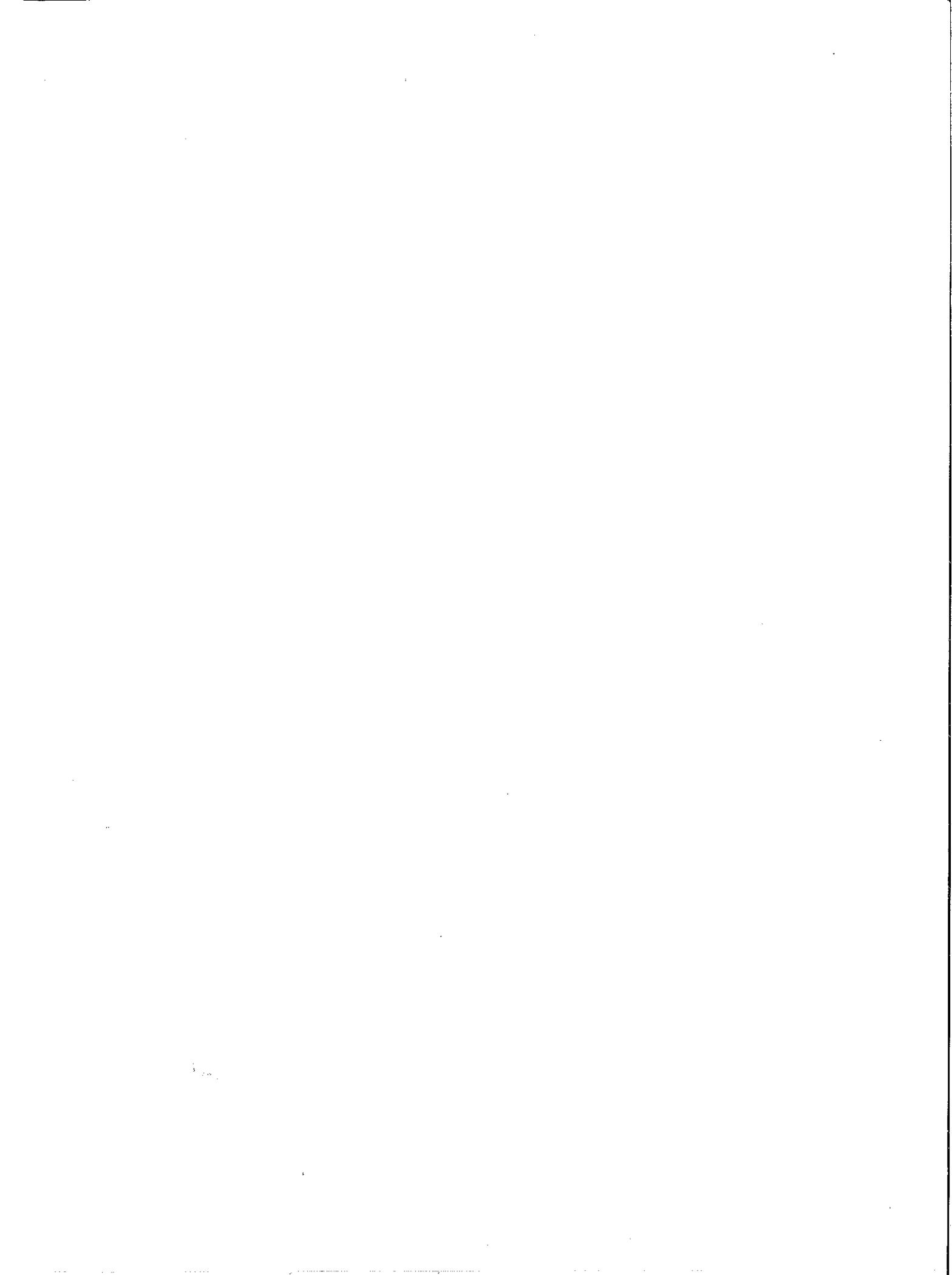
T. gondii

1. Kean BH, Kimball AC, Christensen WN. An epidemic of acute toxoplasmosis. JAMA 1969;208:1002-4.

VIRAL

Hepatitis A

1. Cliver DO. Implications of foodborne infectious hepatitis. Public Health Rep 1966;81:159-65.
2. Gravelle CR, Hornbeck CL, Maynard JE, et al. Hepatitis A: Report of a common-source outbreak with recovery of a possible etiologic agent. II. Laboratory studies. J Infect Dis 1975;131:167-71.
3. Leger RT, Boyer KM, Pattison CP, et al. Hepatitis A: Report of a common-source outbreak with recovery of a possible etiologic agent. I. Epidemiologic studies. J Infect Dis 1975;131:163.



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The State Epidemiologists are the key to all disease surveillance activities and their contributions to this report are gratefully acknowledged. In addition, valuable contributions are made by State Laboratory Directors.

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